

# Calcasieu Estuary Remedial Investigation/Feasibility Study (RI/FS): Baseline Ecological Risk Assessment (BERA)

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## *Appendix I2: Assessment of Risks to Piscivorous Mammals in the Calcasieu Estuary*

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### *Prepared For:*

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### *Under Contract To:*

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## **Appendix I2. Assessment of Risks to Piscivorous Mammals in the Calcasieu Estuary**

### **1.0 Introduction**

Development and industrialization in and around the Calcasieu estuary in southwestern Louisiana in recent decades has led to concerns of environmental contamination in the area. A Remedial Investigation/Feasibility Study (RI/FS) was commissioned to determine the risks posed by environmental contamination to ecological receptors inhabiting key areas of the Calcasieu Estuary. A Baseline Ecological Risk Assessment (BERA) is required to meet this objective. This Appendix is part of the BERA and is conducted in accordance with the procedures laid out by the USEPA in the *Ecological Risk Assessment Guidance for Superfund: Process for Designing and Conducting Ecological Risk Assessment* (USEPA 1997a). Under the eight-step process described by the USEPA for conducting a BERA, a screening ecological risk assessment (SERA) must first be conducted to determine preliminary estimates of exposure and risk.

The SERA for the Calcasieu Estuary (CDM 1999) identified areas of concern (AOCs), contaminants of concern (COCs), and ecological receptors potentially at risk. The SERA findings were revisited in a Baseline Problem Formulation (BPF; MacDonald *et al.* 2001) to yield a refined list of contaminants of concern, areas of interest, and ecological receptors to be considered in the BERA. The Phase II data collection provided more information and, therefore, a better tool to estimate risk at a screening level. Using this information, a conservative, deterministic assessment was conducted and can be found in Appendix G along with a description of the methods used to identify the COCs and AOCs for piscivorous mammals.

This Appendix is organized as follows. Section 1 provides a brief overview of the results of the conservative, deterministic ERA for wildlife described in detail in Appendix G. The AOCs and COCs that screened through the conservative, deterministic assessment for piscivorous mammals are described in this section. Section 1 also includes a description of the conceptual model for piscivorous mammals in the Calcasieu Estuary. A statement outlining the purpose of this assessment concludes Section 1.

Section 2 describes the probabilistic risk assessment methods used to estimate risks of COCs to piscivorous mammals in the Calcasieu AOCs. Section 3 describes the probabilistic risk assessment results and Section 4 identifies the sources of uncertainty that could influence the estimated risks for piscivorous mammals. The final section of this Appendix, Section 5, contains the conclusions regarding risks of COCs to piscivorous mammals in the Calcasieu Estuary.

## **1.1 Deterministic Ecological Risk Assessment Summary**

The methods and results of the deterministic ecological risk assessment are presented in detail in Appendix G. In summary, the deterministic assessment used a conservative approach to estimate risk to piscivorous mammals from chemicals of potential concern (COPCs) in the Bayou d'Inde, Upper Calcasieu River and Middle Calcasieu River Areas of Concern (BI AOC, UCR AOC, MCR AOC, respectively) of the Calcasieu Estuary system. Several reference areas, such as Bayou Bois Connine and Bayou Choupique, were also included in the deterministic assessment to provide a basis for comparison of risks. The deterministic assessment compared potentially attainable high exposures with conservative adverse effects benchmarks to identify which contaminants are a concern to piscivorous mammals and in which

areas of the Calcasieu Estuary system. A risk quotient (total daily intake/effect dose) approach was used to determine if the COPC screened through to the probabilistic ecological risk assessment, using the following decision rules:

- If all RQs were less than 1.0 for all areas of concern for a COPC, the COPC was eliminated from further consideration;
- If RQs were  $\geq 1.0$  for at least one area of concern, but were less than 1.2 times the RQs for reference areas, the COPC was eliminated from further consideration. In these cases, the COPC is unlikely to be causing significant incremental risk in the area of concern over what is occurring in the background; and,
- If RQs were  $\geq 1.0$  for at least one area of concern and were  $\geq 1.2$  times the RQ of the reference area, the COPC screened through to the next phase.

COPCs that were screened through by the SERA are now referred to as contaminants of concern (COCs). Mercury was screened in for all three areas, TCDD-TEQs were screened in for the UCR AOC, MCR AOC and BI AOC, selenium was screened in for the BI AOC and MCR AOC and total PCBs were screened in only in BI AOC. The reference areas were also screened through to the probabilistic risk assessment so that risks in the AOCs could be compared to background risks. Results of the deterministic risk assessment are presented in Table I2-1.

## **1.2 Contaminants of Concern**

The COCs that screened through to the probabilistic risk assessment for piscivorous mammals are mercury, TCDD-TEQs, selenium and total PCBs. These COCs are described below.

### ***Mercury***

Mercury is found in the environment as the metal,  $\text{Hg}^0$ , and as divalent mercuric  $\text{Hg(II)}$  species. In the water column,  $\text{Hg}^0$  is oxidized to  $\text{Hg(II)}$  under acidic conditions.  $\text{Hg(II)}$  undergoes a number of important reactions, one of which is methylation by microbes and adsorption and absorption by biota (Stein *et al.* 1996). Biomethylation occurs both in the sediments, where sulfate-reducing bacteria are the primary methylators of mercury, and in the water column (Winfrey and Rudd 1990). Methylation in the water column also occurs abiotically, mediated by dissolved organic carbon (Weber 1993). Methylmercury may make up as much as 25 percent of the mercury in rivers and lakes (Gilmour and Henry 1991).

Methylmercury is highly soluble in water, extremely mobile, and thus readily enters the aquatic food web. Because methylation is higher under anaerobic conditions, benthic organisms in the anaerobic zones of sediment may be exposed to high methylmercury concentrations. These organisms are consumed by a variety of species, including piscivorous mammals, leading to biomagnification up the food chain. The accumulation of methylmercury in aquatic organisms has been well documented, with concentrations in carnivorous fish 10,000 to >1,000,000 times the concentrations found in ambient waters (Stein *et al.* 1996). Gilmour and Henry (1991) showed that fish from contaminated systems may continue to contain high levels of methylmercury long after inputs to the systems have ceased. Also, the efficient assimilation of the lipophilic methylmercury in fat and muscle and the lack of elimination results in increasing methylmercury concentrations with the age and size of fish and wildlife predators.

This assessment focuses on the risks posed by methylmercury to piscivorous mammals because this species of mercury is more readily bioaccumulated and more toxic to wildlife than is metallic mercury. Further, previous assessments of methylmercury

risks to wildlife have shown that species higher in the aquatic food chain are at particular risk of experiencing adverse effects, including reduced reproduction, impaired growth and development, and death (MacIntosh *et al.* 1994; USEPA 1997b; Moore *et al.* 1999). Piscivorous mammals are high in the food chain and are potentially at high risk of exposure to methylmercury because they consume fish and other aquatic organisms (Environment Canada 2000; NSRL 2002) inhabiting the Calcasieu Estuary.

### ***TCDD-TEQs***

Tetrachlorinated dibenzo-*p*-dioxins-TEQs represent a group of aromatic compounds with similar properties (WHO 1989). The term equivalents refers to a specific group of polychlorinated dibenzo-*p*-dioxin (PCDDs) congeners, co-planar polychlorinated dibenzofuran (PCDFs) congeners and co-planar polychlorinated biphenyl (PCB) congeners. This group has a common structural relationship that includes lateral halogenation and the ability to assume a planar conformation. The planar conformation is important as it leads to a common mechanism of action in many animal species that involves binding to the aryl hydrocarbon (Ah) receptor and elicitation of an Ah receptor-mediated biochemical and toxic response (van den Berg *et al.* 1998; Newsted *et al.* 1995; Safe 1994).

Each of these compounds, while similar in structure and acting at the same receptor, has different potencies, depending on the individual congener. To address these issues and effectively estimate the relative toxicity of these mixtures, a system has been created involving the development and use of toxic equivalency factors (TEFs). This approach is based on the *in vivo* and *in vitro* toxicity of each of the compounds in relation to 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD). TCDD is considered to be the most toxic member of the this class of chemicals (van den Berg *et al.* 1998; Birnbaum and DeVito 1995; Safe 1994) and the toxicity of the others depends on the

degree of chlorination, the chlorination sites, and the ability to achieve a planar form, relative to TCDD. There are a number of assumptions made when using the TEF approach. These include: 1. the congeners are Ah-receptor antagonists and their toxicological potency is mediated by their binding affinity; and 2. no interaction occurs between the congeners and thus the sum of the individual congener effects accounts for the potency of the mixture. The overall effect of these assumptions is a potency estimate or toxic equivalent (TEQ) value. A more detailed discussion of the TEF approach for expressing the toxicity of this class of chemicals is presented in Appendix G.

The environmental degradation and metabolism of the congeners varies due to their unique physical/chemical properties. These can cause substantial differences between the congeners detected in environmental samples and the congener makeup of the original product (van den Berg *et al.* 1998). The majority of these congeners have low solubility, low vapor pressure and high resistance to chemical breakdown, and are, therefore, highly persistent in the environment. They are also highly lipophilic with a high propensity to bind to organic and particulate matter. When released to aquatic systems, the majority of these compounds form associations with dissolved and/or particulate matter in the water column; biodegradation is considered to be a relatively minor fate process in water (NRCC 1981; Howard *et al.* 1991). Aquatic sediments provide a sink for these compounds and may represent long term sources to the aquatic food web (Kuehl *et al.* 1987; Muir 1988; Corbet *et al.* 1983; Tsushimoto *et al.* 1982). As sediments are resuspended and carried downstream, they tend to accumulate in areas where currents are slow and the particles have time to settle.

Organisms may be exposed to TCDD-TEQs through trophic transfer. PCDDs, PCDFs and PCB congeners are highly bioaccumulative substances that increase in

concentration as they are passed up the food chain (i.e., biomagnification). For organisms inhabiting the Lake St. Clair ecosystem, Haffner *et al.* (1994) observed that PCB concentrations increased from 935,000 ng/kg in sediments, to 1,360,000 ng/kg in bivalves, to 7,240,000 ng/kg in oligochaetes, and to 64,900,000 ng/kg in predatory gar pike. Mink are particularly sensitive to PCBs and similar chemicals (Moore *et al.* 1999). Research has found that they accumulate PCBs in their subcutaneous fat at levels 38 to 200 times dietary concentrations, depending on the PCB congener (USEPA 1993). The mammalian predators of the Calcasieu estuary study area would similarly be expected to accumulate PCBs from the prey they consume.

This assessment estimates the risks posed by coplanar congeners to piscivorous mammals because these compounds are expected to biomagnify up the food chain. Further, previous assessments have shown that species higher in the aquatic food chain are at particular risk of experiencing adverse effects, including reduced reproduction, impaired growth and development, and death (Moore *et al.* 1999; Tillitt *et al.* 1996; Heaton *et al.* 1995). Piscivorous mammals are high in the food chain and are potentially at high risk of exposure to coplanar congeners because they consume fish, and other aquatic organisms (Environment Canada 2000; NSRL 2002) that are found in the Calcasieu Estuary system.

### ***Selenium***

The fate of selenium and its compounds in the environment is influenced to a large degree by its oxidation state. The valence states of selenium range from -2 (hydrogen selenide) through 0 (elemental selenium), +2 (selenium dioxide), +4 (selenite) and +6 (selenate). The behavior of various compounds of selenium in the environment is also dependent on ambient conditions including pH, the presence of metal oxides and biological activity (ATSDR 1996; Maier *et al.* 1988).

Elemental selenium is essentially insoluble and will remain inert when released in the environment under anaerobic conditions. Heavy metal selenides and selenium sulfides predominate in acidic soils and soils with high organic matter, and will remain insoluble and immobile in this form (NAS 1976). Selenites and selenates are water soluble and are, therefore, more bioavailable in surface water and water contained in soils (Eisler 2000; ATSDR 1996; Robberecht and Van Grieken 1982). In general, these mobile forms of selenium dominate under aerobic and alkaline conditions. Sodium selenate is one of the most mobile selenium compounds in the environment because of its high water solubility and inability to adsorb onto particulates (NAS 1976). Selenium bioconcentrates and biomagnifies in aquatic food chains from invertebrates to birds (Ohlendorf *et al.* 1986a; 1986b; Lemly 1985; Saiki and Lowe 1987; Saiki *et al.* 1993). Lemly (1985) reported bioconcentration factors of 1,500-1,850 and BAFs of 1,746-3,975 for selenium in freshwater species. Concentrations of selenium in river otter and raccoon have been measured (wet weight) in various organs ranging from 0.2 to 2.8 mg Se/kg (Wren 1984). These studies demonstrate that selenium has the potential to biomagnify up the food chain and accumulate in piscivorous mammals.

This assessment focuses on the risks posed by selenium to piscivorous mammals because this substance is expected to biomagnify up the food chain. Selenium bioconcentrates in aquatic food chains from invertebrates to birds with diet being identified as the primary source for fish and piscivorous birds having the highest body burdens among birds (Eisler 2000). Piscivorous mammals are high in the food chain and are potentially at high risk of exposure to selenium because they consume, fish, and other aquatic organisms (Environment Canada 2000; NSRL 2002) that are found in the Calcasieu Estuary system.

***Total PCBs***

Polychlorinated biphenyls (PCBs) is the generic term applied to a group of 209 chlorinated organic compounds that have similar molecular structures and properties. The majority of PCBs congeners tend to have low solubilities, low vapor pressure and high resistance to chemical breakdown. Due to chemical stability, PCBs are highly persistent in the environment.

PCBs are persistent and highly lipophilic substances with low water solubility and a high propensity to bind to organic and particulate matter. In bulk releases to aquatic compartments, these substances will tend to remain as a non aqueous phase liquid and settle to the bottom of the water body. Here, PCBs will gradually adsorb to organic and particulate matter and remain sequestered in sediment layers. Exposure to PCBs from this compartment occurs as a result of benthic organisms ingesting sediments during foraging and when sediments are stirred and PCB-laden particles resuspended in the water column. As sediments are resuspended and carried downstream, they tend to accumulate in areas where currents are slow and the particles have time to settle.

Predatory organisms may be exposed to chemical contaminants through trophic transfer. Organisms lower in the food chain may ingest and accumulate a substance, which is then passed on when they are consumed by higher food chain predators. Benthic communities are at the highest risk of direct exposure to PCBs. Benthic invertebrates will be exposed to PCBs through direct contact with interstitial pore water, ingestion of sediment particles, and ingestion of organisms that have also been exposed to contaminants. Pelagic organisms in the Calcasieu estuary will be exposed to PCBs through dermal and gill contact with surface waters; ingestion of water, suspended sediment, and organic matter; ingestion of sediment for bottom-feeding fish; and ingestion of other benthic and pelagic organisms. Uptake of PCBs by fish

occurs mainly through the gills and the gastrointestinal tract (Shaw and Connell 1984). Most PCB accumulation in top fish predators can be attributed to the food pathway (Thomann 1989). Other species, such as amphibians, are also exposed to PCB-contaminated surface waters. Insectivorous, carnivorous, and piscivorous birds and mammals that reside, or partially reside, in the estuary are exposed to PCBs principally through diet and trophic transfer. PCBs are highly bioaccumulative substances that increase in concentration as they are passed up the food chain. For organisms inhabiting the Lake St. Clair ecosystem, Haffner *et al.* (1994) noted that PCB concentrations increased from 0.935 mg/kg in sediments, to 1.36 mg/kg in bivalves, to 7.24 mg/kg in oligochaetes, and to 64.9 mg/kg in predatory gar pike. PCBs have also been observed to biomagnify in several birds (Senthilkumar *et al.* 2001; Borga *et al.* 2001). The avian and mammalian predators of the Calcasieu estuary study area would similarly be expected to accumulate PCBs from the prey they consume.

This assessment focuses on the risks posed by total PCBs to piscivorous mammals because PCBs are expected to biomagnify up the food chain. Further, previous assessments of PCBs risks to wildlife have shown that species higher in the aquatic food chain are at particular risk of experiencing adverse effects, including reduced reproduction, impaired growth and development, and death (Moore *et al.* 1999). Piscivorous mammals are high in the food chain and are potentially at high risk of exposure to PCBs because they consume, fish, and other aquatic organisms (Environment Canada 2000; NSRL 2002) that are found in the Calcasieu Estuary system.

### 1.3 Receptors of Concern

Thorough observations of the study area led to the identification of a number of mammalian species including bats (Order Chiroptera), rabbit (*Sylvilagus sp.*), raccoon (*Procyon lotor*), eastern fox squirrel (*Sciurus niger*), nutria (*Myocastor coypus*), river otter (*Lutra canadensis*), white-tailed deer (*Odocoileus virginianus*), and dolphins (Delphinidae; ChemRisk 1996). The number of piscivorous mammalian species that feed on the aquatic prey and have the potential to occur in the study area is quite limited.

The exposure assessment for piscivorous mammals exposed to COCs will be based on a hypothetical receptor that incorporates many of the characteristics typical of this receptor group. The characteristics of this hypothetical receptor are based upon of two species of aquatic-dependent piscivorous mammals potentially found in the Calcasieu Estuary: mink and river otter. These animals are opportunistic piscivores that may consume aquatic invertebrates and fish as parts of their diets. The following sections review the life histories and foraging behaviors of these two species. This information is then used to develop the life history and foraging behavior of the hypothetical receptor that will be used in this assessment.

#### **Mink (*Mustela vison*)**

Mink are semi-aquatic mammals. Males are larger than females. Males are between 33 and 43 cm in length with an 18 to 23 cm tail. Females, on the other hand, range from 30 to 36 cm in length with a 13 to 20 cm tail (USEPA 1993). Mink typically weigh between 0.55 and 1.1 kg with males weighing almost twice as much as females (Birks and Dunstone 1985; Mitchell 1961). Mink occur throughout North America, with the exception of the southwest United States (Lowery 1974; Choate *et al.* 1994). In Louisiana, mink are found statewide.

In Louisiana, mink are particularly numerous in tupelo-gum swamps, in the freshwater to brackish coastal marshes, along wooded streams, and on the edges of lakes. Mink are never found far from water. Habitat preferences include irregular shorelines covered with brush and woody cover. Fallen debris on shorelines is also useful, as it creates excellent den habitat (USEPA 1993). The shape of mink home range depends on habitat type. Riverine home ranges are generally linear while those in marsh habitats tend to be more circular (Eagle and Whitman 1987). Mean home range size for adult mink ranges from about 2-3 kilometers of stream or river (USEPA 1993). Population densities range from about 0.01 to 0.1 mink per hectare or one mink per two kilometers of shoreline. Mink will defend 1 to 4 km of shoreline with scent markings and physical aggression (USEPA 1993).

Mink are predominantly nocturnal feeders and can be described as opportunistic. They will feed on a variety of prey, depending on the season and prey abundance. The majority of hunting takes place along shorelines or in emergent vegetation. As a result of their smaller size, female mink do not consume large prey, such as muskrats and rabbits that males consume (Birks and Dunstone 1985; USEPA 1993). Prey that both males and females consume include aquatic animals such as fish, amphibians and crustaceans, as well as terrestrial animals like small mammals, birds, reptiles, and insects (USEPA 1993). Mink are almost strictly carnivorous with only a limited amount of plant material reported in their diet (Proulx *et al.* 1987). Feeding habitats can be affected by water level. During high water levels mink feed more on crayfish and voles (Proulx *et al.* 1987). When water levels are low, their diet switches to aquatic birds, muskrats, and even ducklings. Winter also affects mink diet; during these months, fish become a more important food source (USEPA 1993). Mink tend to feed on slow-moving bottom fish rather than on the faster mid-stream salmonids (Eagle and Whitman 1987). Fish species captured by mink in rivers and streams in lower Michigan and in New York were under 15-18 cm in length

(Hamilton 1959; Alexander 1977). Using the year round study conducted by Alexander (1977), USEPA (1995) estimated that the average trophic level of the prey of mink is 2.9 based on size of fish. They also estimated that the proportion of the diet taken from aquatic food webs ranged from 75-90 percent, depending on habitat. Other studies have estimated that aquatic food constituted a lower proportion of diet. These studies measured dietary composition by percent frequency of occurrence, however, which may not reflect percent biomass well (USEPA 1995).

### **River Otter (*Lutra canadensis*)**

River otter are a long-bodied, short-legged, semi-aquatic animal. Males range in weight from 5 to 10 kg and females from 4 to 7 kg (Melquist and Hornocker 1983). Body length ranges from 66 to 76 cm with a 30 to 43 cm tail. The river otter occurs throughout most of Canada and the continental United States, except for the southwestern United States (Lowery 1974; Choate *et al.* 1994). It occurs throughout the state of Louisiana where suitable habitat is available along streams and river and in coastal marshes.

Otters spend most of their time in and near rivers, creeks, bayous, and lakes, especially those bordered by timber. River otters prefer habitat close to lakes, marshes, streams, and seashores. When selecting habitat, abundance of food is a primary consideration. River otters den in banks and hollow logs (USEPA 1993). The shape of the home range is similar to mink and varies by habitat type. River otter home range size is determined by the area needed to meet the demands of foraging and reproduction. Mean home range size for adult size otter are approximately 30 km of shoreline (Melquist and Hornocker 1983). It is common to find one river otter for every km to one otter for every 10 km of shoreline. Males tend to range more than females, with lactating females ranging the least (USEPA 1993).

River otter tend to be piscivorous. However, they are opportunistic feeders and will also prey on frogs, turtles, snakes and aquatic invertebrates such as crayfish and crabs. Occasionally, they eat birds, rats, and mice. River otters can capture adult trout, salmon, perch and pike. However, because these prey are fast swimmers and difficult for otter to capture, they comprise a small part of their diet on a yearly basis (Lauhacinda 1978). River otters may probe the bottom of ponds or streams for invertebrates and thus ingest sediment and other debris in the process (USEPA 1993). The USEPA (1995) reviewed available field studies on river otter diet. They found that river otter diets varied markedly depending on the season. For example, where fish may not be as readily available in some regions during the winter months due to ice cover, river otter diets were more dependent on other organisms. Because river otters are fairly large mammals and are opportunistic in their feeding habits, fish size can vary greatly from 2 to 50 cm (Melquist and Hocnocker 1983). However, most fish captured are small and less than 15 cm in length (Hamilton 1961; Lagler and Ostenson 1942; Alexander 1977). Greer (1956) and Chanin (1981) determined that fish prey size captured by otter ranged from less than 15 cm (60%), to between 15-25 cm (30%) and greater than 25 cm (5%). The trophic level of prey items consumed by the northern river otter vary depending on the source of the prey. USEPA (1995) estimated that the average aquatic trophic level for otter throughout the year is likely to be between 2.7 and 3.2 in most regions of the country.

### **Hypothetical Piscivorous Receptor of Concern**

The hypothetical piscivorous mammal receptor for this assessment is based upon the behavior and characteristics described for mink and river otter, as follows:

- The receptor body weight is approximately equal to the average of the two species considered above. The coefficient of variation (CV) for body weight is also approximately equal to the CV of 10% for adults of

piscivorous mammals. To ascertain whether smaller piscivorous mammals might be at greater risk because of their higher metabolic rate, we conducted a “what if” analysis using a receptor body weight typical of mink with a CV of 10%;

- The hypothetical receptor is assumed to have a relatively small foraging range with high site fidelity and no territoriality. Receptors will forage exclusively within the BI AOC, MCR AOC, UCR AOC, or reference areas. Each foraging area range assumes sufficient habitat quality and prey abundance;
- Because the hypothetical receptor is piscivorous and opportunistic, the diet of the hypothetical receptor is assumed to consist almost entirely of fish, which is the dominant part of the diet of mink and river otter. However, invertebrates are also part of the diet for the receptor;
- Most of the piscivorous mammals identified in the area are opportunistic in terms of habitat as well as diet. As long as there is water, sufficient food, and suitable shelter most of these animals could occur in Calcasieu Estuary habitats; and,
- The hypothetical receptor is assumed to be resident year-round in each of the Calcasieu Estuary areas. The temporal scale for this assessment is long term because: (1) contaminant levels are unlikely to exhibit high temporal variability; and, (2) chronic toxicity typically occurs at much lower levels than acute toxicity.

## 1.4 Conceptual Model

The conceptual model illustrates the relationships between sources and releases of COCs, their fate and transport, and the pathways through which COCs reach piscivorous mammals and exert potential adverse effects. The model enhances the level of understanding regarding the relationships between human activities and ecological receptors at the site under consideration. In so doing, the conceptual model provides a framework for predicting effects on ecological receptors and a template for generating risk questions and testable hypotheses (USEPA 1997a; 1998). The conceptual site model developed for the Calcasieu Estuary is described in greater detail in Chapter 7 of the BPF. The conceptual model summarizes information on the sources and releases of COCs, the fate and transport of these substances, the pathways by which ecological receptors are exposed to the COCs, and the potential effects of these substances on the ecological receptors that occur in the Calcasieu Estuary. In turn, this information is used to develop a series of risk hypotheses that provide predictions regarding how ecological receptors will be exposed to and respond to the COCs.

Piscivorous mammals are exposed to a number of COCs in the Calcasieu Estuary system and the deterministic risk assessment (Appendix G) identified those COCs that pose potential risks to these animals. Specifically, piscivorous mammals are at greatest risk from mercury, TCDD-TEQs, selenium and total PCBs in the Calcasieu Estuary. These substances are persistent and bioaccumulative and are available for uptake by piscivorous mammals, primarily through the food chain. The Phase II sampling program provided data identifying substantial tissue residues of these substances in fish and aquatic invertebrates, which are prey items of many piscivorous mammals. Other routes of exposure, including inhalation, water consumption and sediment

ingestion have been excluded from this assessment as their contribution to overall exposure is likely negligible.

## **1.5 Assessment Endpoints**

An assessment endpoint is an ‘explicit expression of the environmental value that is to be protected’ (USEPA 1997a). The selection of assessment endpoints is an essential element of the overall ERA process because it focuses assessment activities on the key environmental values (e.g., reproduction of piscivorous mammals) that could be adversely affected by exposure to environmental contaminants. Assessment endpoints must be selected based on the ecosystems, communities, and species that occur, have historically occurred, or could potentially occur at the site (USEPA 1997a).

To support the identification of key assessment and measurement endpoints for the Calcasieu Estuary BERA, the United States Environmental Protection Agency (USEPA) convened a BERA workshop in Lake Charles, LA on September 6 and 7, 2000. The workshop participants included representatives of the USEPA, United States Geological Service (USGS), National Oceanic and Atmospheric Administration (NOAA), Louisiana Department of Environmental Quality (LDEQ), United States Fish and Wildlife Service (USFWS) and CDM Federal. The workshop was designed to enable participants to articulate the goals and objectives for the ecosystem (i.e., based on the input that had been provided by the community in a series of public meetings), to assess the state of the knowledge base, to define key issues and concerns, and to identify the chemicals and areas of potential concern in the study area. This workshop provided a basis for refining the candidate assessment endpoints that had been proposed based on the results of the SERA (CDM 1999). Workshop

participants also identified a suite of measurement endpoints that would provide the information needed for evaluating the status of the assessment endpoints (MacDonald *et al.* 2000a).

Aquatic-dependent mammals are linked to aquatic ecosystems as a result of their reliance on aquatic organisms for food. These species can be classified based on their feeding habits into two main groups: omnivorous mammals (i.e., species that eat a wide variety of plants and animals, including aquatic organisms) and piscivorous mammals (i.e., species that eat fish). Due to their reliance on aquatic organisms for food, it is important to evaluate the effects of environmental contaminants on this group of ecological receptors. The assessment endpoint for the assessment of risk to piscivorous mammals in the Calcasieu estuary is survival, growth and reproduction of piscivorous mammals.

Although mammals can be exposed to environmental contaminants through dermal contact with contaminated surface water or sediments (i.e., dermal exposure) or consumption of contaminated surface water, the bulk of their exposure is associated with the consumption of contaminated prey items. This is especially true for persistent and bioaccumulative COCs. Therefore, it is important to evaluate the effects of contaminated prey items on the survival and reproduction of mammals.

## **1.6 Measurement Endpoints**

A measurement endpoint is defined as ‘a measurable ecological characteristic that is related to the valued characteristic selected as the assessment endpoint’ and it is a measure of biological effects (e.g., mortality, reproduction, growth; USEPA 1997a). Measurement endpoints are frequently numerical expressions of observations (e.g.,

toxicity test results, community diversity measures) that may or may not be compared to similar observations at a control and/or reference site.

A single measurement endpoint will be used to evaluate the risks to piscivorous mammals. The potential for adverse effects on piscivorous mammals will be evaluated using the comparison of prey tissue data and the results of laboratory studies. Specifically, the data on the concentrations of contaminants measured in fish (i.e., primarily < 15 cm in length) and aquatic invertebrates (< 12.5 cm in length) will be used. These data will be compiled by geographic area within the estuary (based on the diet and foraging range of a hypothetical mammal species), incorporated into a daily intake exposure model, and compared to appropriate toxicity values for survival and reproduction of piscivorous mammals.

## **1.7 Risk Hypothesis and Questions**

The following risk hypothesis was developed to identify the key stressor-effect relationships that will be evaluated in the probabilistic ecological risk assessment:

Based on the physical-chemical properties (e.g.,  $K_{ow}$ s) of the bioaccumulative contaminants of concern, the nature of the food web in the Calcasieu Estuary, and the effects that have been documented in laboratory studies, mercury, TCDD-TEQs, selenium and total PCBs released into surface waters will accumulate in the tissues of aquatic organisms to levels that adversely affect the survival, growth, and/or reproduction of piscivorous mammals.

To assess ecological risks, the assessment endpoint must be linked to the measurement endpoint by risk questions. In this study, the investigation to assess the risks of COCs to mammals was designed to answer the following risk questions:

- Are the levels of contaminants in the tissues of prey species of piscivorous mammals in the Calcasieu Estuary sufficient to cause adverse effects to survival, growth or reproduction?
- If yes, what are the probabilities of effects of differing magnitude for survival, growth and/or reproduction of piscivorous mammals?

The linkages between the assessment endpoint and the measurement endpoints are articulated in greater detail in Table A1-21 of the Baseline Problem Formulation (MacDonald *et al.* 2001).

## **1.8 Purpose of Appendix**

The purpose of this assessment is to test the above risk hypothesis by characterizing the risks posed to the piscivorous mammalian community associated with exposure to the COCs identified in Appendix G.

## **2.0 Methods**

A step-wise approach was used to assess the risks to the mammalian community posed by the COCs in the Calcasieu Estuary. The four main steps in this process included:

1. Collection, evaluation, and compilation of the relevant data on the concentrations of COCs in prey items and sediment in the Calcasieu Estuary;
2. Assessment of exposure of mammals to COCs (Figure I2-1);
3. Assessment of the effects of COCs on mammals (Figure I2-2); and,
4. Characterization of risks to piscivorous mammals (Figure I2-3).

Each of these steps is described in this Appendix. The results of the deterministic assessment were briefly reviewed in Section 1.1. For details of this assessment, see Appendix G.

## **2.1 Collection, Evaluation, and Compilation of Data**

Information on chemical levels in tissues of prey of piscivorous mammals were collected in two phases, termed the Phase I and Phase II sampling programs. The Phase I program results indicated that the detection limits for many of the COCs in tissues were orders of magnitude above corresponding benchmarks. Therefore, the Phase I results for tissues were not considered in this assessment. The methods used to collect the tissue samples in the Phase II program, quantify the levels of COCs, evaluate the reliability of the data, and compile the information in a form that would support the BERA are described in the following sections.

***Sample Collection of Tissues*** - More than 600 tissue samples were collected at sites located throughout the estuary between October, 2000 and November, 2000.

Biota tissue samples were collected in three AOCs in the estuary (Upper and Middle Calcasieu Rivers and Bayou d'Inde areas of concern) and in the reference areas (Bayou Bois Connine, Bayou Choupique, Grand Bayou, Johnson Bayou and Willow

Bayou). There were also a number of sub-areas within the AOCs from which samples were taken. The USEPA Region V FIELDS tools were used to randomly select coordinates (i.e., latitude and longitude) for the assigned number of primary sampling stations and alternate sampling stations (i.e., which were sampled when it was not possible to obtain samples from the primary sampling stations). In the field, each sampling station was located with the aid of navigation charts and a Trimble differentially-corrected global positioning system (GPS). Using standard statistical power analysis methods, an evaluation of previously collected data was completed to determine the number of samples to be collected within each area and sub-area. In addition, samples of fiddler crabs and *Rangia* were also collected in October 2001.

The methods used to collect, handle, and transport the tissue samples are described in CDM (2000a; 2000b; 2000c; 2000d; and 2000e). Briefly, fish and invertebrate species were collected by hook and line, hand collection and netting. Minnows and other small bait species were collected using legal cast nets, minnow traps, dip nets and bait seines in accordance with the Louisiana Department of Wildlife and Fisheries. Each sample was wrapped in aluminum and put in a Ziploc® bag. All samples were kept frozen and shipped to laboratories in coolers on dry ice.

***Chemical Analyses of Tissues*** - Chemical analysis of the tissue samples was conducted at various contract laboratory program (CLP) and subcontract (non-CLP) analytical laboratories, including USEPA Region VI Laboratory, USEPA Region VI CLP laboratories, Olin Contract laboratories, Texas A&M University laboratories, ALTA laboratories, AATS laboratories and EnChem laboratories. Upon receipt at the laboratory, tissue samples were held in freezers until analysis.

All tissue samples were analyzed for total target analyte list (TAL) metals, target compound list (TCL) semi-volatile organic compounds (SVOCs) and TCL pesticides.

Total metals were quantified using the SW6010B method. Polycyclic aromatic hydrocarbons and/or other semi-volatile organic compounds were quantified using the SW8270C method. Methods SW8081A and SW8082 were used to quantify pesticides. Twenty percent of the tissue samples were analyzed for PCB congeners and dioxins/furans. EPA Method SW1668 was used to quantify PCB congeners and SW8290 was used for dioxins/furans.

EnChem laboratories used additional analytical methods to quantify mercury, polycyclic aromatic hydrocarbons (PAHs), pesticides and dioxins and furans. Methods 1631MOD and 1630MOD were used to quantify mercury and methylmercury, respectively. PAHs were quantified using Method 8270C-SIM. Method SW8082 and AXYS Method CL-T-1668A/Ver.3 were used to quantify pesticides. Dioxins and furans were quantified using AXYS Method DX-T-8290/Ver.2.

***Data Validation and Verification*** - All of the data sets generated during the course of the study were critically reviewed to determine their applicability to the assessment of risks to the biotic community in the Calcasieu Estuary. The first step in this process involved validation of the tissue chemistry data. Following translation of these data into database format, the validated data were then further evaluated to ensure the quality of the data used in the risk assessment. We were unable to confirm tissue data results against the original source.

***Database Development*** - To support the compilation and subsequent analysis of the information on biota in the Calcasieu Estuary, a relational project database was developed in MS Access format. All of the tissue chemistry data compiled in the database were georeferenced to facilitate mapping and spatial analysis using geographic information system (GIS)-based applications (i.e., ESRI's ArcView and

Spatial Analyst programs). The database structure made it possible to retrieve data in several ways, including by data type (i.e., chemistry vs. toxicity), by stream reach (i.e., Upper Bayou d'Inde vs. Lower Bayou d'Inde), by sub-reach (i.e., Upper Bayou d'Inde-1 vs. Upper Bayou d'Inde-2), and by date (i.e., Phase I vs. Phase II). As such, the database facilitated a variety of data analyses.

## **2.2 Probabilistic Ecological Risk Assessment**

Monte Carlo analysis is an increasingly widely used approach to probabilistic risk assessment (USEPA 1997c; 1999). It is used to propagate uncertainty associated with the variability of input variables, as well as any incertitude associated with how to parameterize input distributions. In this assessment, we use probability bounds analysis to determine the relative contributions of incertitude and variability to exposure estimates (see Chapter 9 of MacDonald *et al.* 2001 for more information on the uncertainty analysis approaches used here).

Monte Carlo analysis requires the specification of the statistical distributions of each of the input variables and their interdependencies as measured by correlations. Computer software such as Crystal Ball is used to 'sample' from these distributions and, via the exposure model equation, compute an exposure distribution. This process is repeated many times so as to build up a histogram that serves as the estimate of the full distribution of exposures (explicitly including the tail risks of extreme exposure).

Probability bounds analysis is an exact numerical approach (not based on simulation) that takes as input the same probability distributions used in Monte Carlo simulation, or, when they are difficult to specify precisely, bounds on these distributions (Ferson

*et al.* 2002). The method then rigorously computes bounds on the cumulative distribution function. The spread between the bounds of an input or output set of distributions corresponds directly to the amount of uncertainty we have about how to describe the variable. Probability bounds analysis is also useful when independence assumptions are untenable, or when sparse empirical data make it difficult to quantify the correlations among variables.

### **2.2.1 Exposure Characterization**

We estimate exposure of piscivorous mammals to methylmercury, TCDD-TEQs, selenium and total PCBs via a daily intake model that considers the dietary ingestion route of exposure. Piscivorous mammals are unlikely to use the saline waters of BI AOC as a source of drinking water and the inhalation route of exposure has been shown to be an insignificant source of hydrophobic contaminants in previous assessments of the risks of these substances to aquatic-dependent wildlife (e.g., Moore *et al.* 1999). Sediment ingestion was also considered as a possible route of exposure, however, deterministic analysis indicated that the contribution of sediment intake to overall exposure of COCs was insignificant. Therefore, the exposure model used in this assessment only includes the ingestion of food items as an exposure route. This exposure assessment assumes that the hypothetical receptor is present year round in each of the identified Areas of Concern.

The temporal scale for this assessment is long term because: (1) levels of mercury, TCDD-TEQs, selenium and total PCBs are unlikely to exhibit high temporal variability; and, (2) chronic toxicity occurs generally at lower levels than acute toxicity. The spatial scale of this assessment is considered to be consistent with home ranges reported for piscivorous mammals (USEPA 1993). The foraging area for the

hypothetical receptor is set to 1 to 15 km of shoreline 25-m wide which easily fits within each of the identified Areas of Concern.

The exposure model calculates the total daily intake of methylmercury, TCDD-TEQs, selenium and total PCBs associated with the ingestion of food. Chemical assimilation efficiency terms are not included in the exposure equation because the efficiencies of chemical adsorption in wild animals following ingestion will likely be similar to the efficiencies in laboratory animals exposed to the substances in toxicity studies. Thus, the chemical assimilation efficiency terms will cancel out when the exposure and effect estimates are combined to estimate risk.

The exposure model is adapted from USEPA (1993) and is represented as:

$$TDI = \frac{FMR \times \sum_{i=1}^n (C_i \times P_i)}{AE_i \times GE_i} \quad (1)$$

where:

$TDI$  = total daily intake of the chemical from diet (mg/kg bw/day),

$C_i$  = concentration of the chemical in the  $i^{\text{th}}$  prey species (mg/kg),

$P_i$  = proportion of the  $i^{\text{th}}$  prey species in the diet,

$FMR$  = normalized free metabolic rate of the wildlife receptor (Kcal/kg bw/day),

$GE_i$  = gross energy of  $i^{\text{th}}$  prey species (Kcal/kg prey),

$AE_i$  = assimilation efficiency of the  $i^{\text{th}}$  prey species (unitless),

Each input variable is described in detail below, including the parameterizations for the Monte Carlo analysis and the probability bounds analysis.

### **2.2.1.1 Selection Criteria for Input Distributions**

The distributions and distribution parameters used in the exposure analyses are summarized in Table I2-2 and Table I2-3. Input distributions were assigned as follows: lognormal distributions for variables that are positively skewed with a lower bound of zero and no upper bound; beta distributions for variables bounded by zero and one (e.g., prey assimilation efficiency); and, normal distributions for variables that are symmetric and not bounded by one (e.g., body weight). The lognormal distribution is often used to provide good representations for physical quantities constrained to being non-negative, and that are positively skewed, such as contaminant concentrations, stream flows, or magnitudes of accidents (Small 1990). Ott (1995) provides an extensive discussion of the theoretical reasons for why contaminant concentrations in the environment are expected to be lognormally distributed. The beta distribution provides a flexible means of representing variability over a fixed range, such as zero to one (Small 1990). The beta distribution can take on a wide variety of shapes between the fixed endpoints and this flexibility has led to its empirical use in diverse applications. The normal distribution arises in many cases because of the central limit theorem which results in a normal distribution for additive quantities such as body weights (Small 1990). The normal distribution can often be used for variables that are non negative, as long as coefficients of variation (CV) are small. This is because many distributions converge to a normal distribution as CVs become small. With most random number generators, it is impossible to obtain numbers more than five standard deviations from the mean. Thus, as long as the CV is less than 0.2, there is no concern for selecting negative values for non-negative variables.

### **2.2.1.2 Input Distributions**

#### ***Body Weight (BW)***

Although body weight data are not used in the exposure model directly, they are a required variable in allometric models used to estimate the free metabolic rate. For this assessment, we used body weights that represent an average-sized hypothetical receptor and a small-sized hypothetical receptor.

For the Monte Carlo analysis, the average body weight of mink and river otter was used (i.e., 3.96 kg). Because the feeding guild encompasses species with widely varying body weights, the calculation of the standard deviation of the mean body weight would have yielded an unduly wide distribution. Instead, we adopted a coefficient of variability (CV) of 10%, which is typical of the body weight distribution for many mammals (Mitchell 1961; Lauhachinda 1978; Melquist and Hornocker 1983; Birks and Dunstone 1985). The application of the adopted CV yielded a standard deviation of 0.396.

We also repeated the Monte Carlo analysis with a mean body weight of 0.608 kg (standard deviation equal to 0.0669). This body weight is representative of the smallest mammal in the guild, mink. Mink, which are smaller animals than otter, tend to have higher metabolic rates (when normalized to body weight) and, as a result, may be at higher risk of exposure.

Body weights were assumed to be distributed normally. The entire proportion of uncertainty in this variable is likely due to variability, with little incertitude. Thus, probability bounds were not established for this input variable.

***Free Metabolic Rate (FMR)***

The energy requirements of piscivorous mammals have not been empirically determined. Nagy (1987) derived an allometric equation for estimating the metabolic rate of free-living mammals using the general equation:

$$FMR \text{ (kJ / d)} = a \cdot BW(g)^b \quad (2)$$

For both the Monte Carlo and the probability bounds analyses, FMR for piscivorous mammals was estimated with a probabilistic approach wherein distributions were derived for each of the input variables (body weight [BW], a, b) and combined according to the above equation. The slope (a) and power (b) distributions were based on the error statistics reported in Nagy (1987), assuming an underlying normal distribution for each. For non-herbivores, log a had a reported mean of 0.412 and a standard error of 0.058, and b had a reported mean of 0.862 and a standard error of 0.026 (Nagy 1987). The body weight (BW) distribution was described above.

***Proportion of Prey Items in Diet (P<sub>i</sub>)***

For the purposes of this assessment, the hypothetical piscivorous mammal receptor is assumed to have a diet that is 60% for group 1a, 2a and 2b fish, 20% group 3a and 3b fish, 10% group 4a and 4b fish, and 10% for 1a, 1b and 2a invertebrates. These values are point estimates with no distributions.

***Gross Energy of Prey (GE<sub>i</sub>)***

Gross energies of fish and invertebrates, which are dietary food items consumed by piscivorous mammals, were available from the literature. The gross energies of these organisms were reported as follows: fish = 1200 Kcal/kg (standard deviation = 240; Thayer *et al.* 1973); crabs = 1000 Kcal/kg (standard deviation = 210; Thayer *et al.* 1973); and, shrimp = 1100 Kcal/kg (standard deviation = 240; Cummins and

Wuycheck 1971). For aquatic invertebrates consumed by piscivorous mammals, the mean gross energy was set to 1050 Kcal/kg (standard deviation = 225) in the Monte Carlo analysis. For fish, the distribution parameters measured by Thayer *et al.* (1973) were used. The distribution for these variables was assumed to be normal. Incertitude was considered low for these input variables because: (1) sufficient experimental data were available to confidently estimate the mean and standard deviation; (2) the variable is easily measured and thus measurement error is low; and, (3) there appears to be little difference in the gross energies of different invertebrate species or different fish species. Therefore, probability bounds were not derived for this variable.

#### ***Assimilation Efficiency of Prey (AE<sub>i</sub>)***

Assimilation efficiencies of mammals consuming fish and insects were reported to be 91% (estimated standard deviation = 9%) and 87% with a standard deviation of 4.9, respectively (USEPA 1993; Grodzinski and Wunder 1975; Barrett and Stueck 1976). A beta distribution was assumed for this variable with the following parameterization for fish and insects, respectively: alpha = 65, beta = 6.7, and scale = 1.0; alpha = 65, beta = 10, and scale = 1. Incertitude was considered low for this input variable because: (1) the variable is easily measured and thus measurement error is low; and, (2) there appears to be little difference in the assimilation efficiencies of different prey species consumed by mammals. Therefore, probability bounds were not derived for this variable.

#### ***Concentration of Methylmercury in Fish (C<sub>f</sub>)***

Total mercury concentrations were used as a surrogate for methylmercury when methylmercury concentrations were not available. In fish tissues, methylmercury accounted for close to 100% of total mercury (Bloom 1992).

*Bayou d'Inde Area of Concern (BI AOC)*

A number of group 1, 2a and 2b fish species including gulf killifish, gulf menhaden and spot, group 3a and 3b fish species including striped mullet and spotted seatrout, and group 4a and 4b fish species including black drum and gizzard shad were sampled in the BI AOC of the Calcasieu Estuary. These were analyzed for total mercury concentrations in fish. The mean was derived by fitting the data to a lognormal distribution using Crystal Ball 2000 (Decisioneering 2000). The fitted mean for group 1, 2a and 2b fish was 0.189 mg/kg bw/day with a standard deviation of 0.193. The fitted mean for 3a and 3b fish was 0.134 mg/kg ww with a standard deviation of 0.0511 and group 4a and 4b had a fitted mean of 0.169 mg/kg ww with a standard deviation of 0.122. During long exposures, piscivorous mammals will spatially and temporally average their exposures. To represent this averaging, we used a bootstrapping process to sample from the mercury in fish distributions over a period of 160 days. Thus, piscivorous mammals were assumed to forage over 160 days, each day consuming group 1, 2a and 2b fish having a lognormal distribution with a mean of 0.189 mg/kg ww and standard deviation of 0.193, group 3a and 3b fish having a lognormal distribution with a mean of 0.134 mg/kg ww and standard deviation of 0.0511, and group 4a and 4b fish having a lognormal distribution with a mean of 0.169 mg/kg ww and standard deviation of 0.122. The resulting grand mean and grand standard deviation for the 160 days was 0.188 mg/kg ww and 0.00536 for group 1, 2a and 2b fish, 0.133 mg/kg ww and 0.00174 for group 3a and 3b fish, and 0.168 mg/kg ww and 0.00350 for group 4a and 4b fish, respectively. This variable was assumed to be lognormally distributed.

For the probability bounds analysis, the Land statistic was used to determine the lower and upper 95% confidence limits on the mean. The resulting values were used to parameterize a lognormal distribution for each fish prey group.

*Upper Calcasieu River Area of Concern (UCR AOC)*

A number of group 1, 2a and 2b fish species including gulf killifish, gulf menhaden and spot, group 3a and 3b fish species including striped mullet and spotted seatrout, and group 4a and 4b fish species including black drum and gizzard shad were sampled in the UCR AOC of the Calcasieu Estuary. These were analyzed for total mercury concentrations in fish. The fitted mean for group 1, 2a and 2b fish was 0.0449 mg/kg ww with a standard deviation of 0.0294. The fitted mean for 3a and 3b fish was 0.0607 mg/kg ww with a standard deviation of 0.0522 and group 4a and 4b fish had a fitted mean of 0.0761 mg/kg ww with a standard deviation of 0.0691. During long exposures, piscivorous mammals will spatially and temporally average their exposures. To represent this averaging, we used a bootstrapping process to sample from the mercury in fish distribution over a period of 160 days. Thus, piscivorous mammals were assumed to forage over 160 days, each day consuming group 1, 2a and 2b fish having a lognormal distribution with a mean of 0.0449 mg/kg ww and standard deviation of 0.0294, group 3a and 3b fish having a lognormal distribution with a mean of 0.0607 mg/kg ww and standard deviation of 0.0522, and group 4a and 4b fish having a lognormal distribution with a mean of 0.0761 mg/kg ww and standard deviation of 0.0691. The resulting grand mean and grand standard deviation for the 160 days was 0.0445 mg/kg ww and 0.000995 for group 1, 2a and 2b fish, 0.0607 mg/kg ww and 0.00191 for group 3a and 3b fish, and 0.0761 mg/kg ww and 0.00255 for group 4a and 4b fish, respectively. This variable was assumed to be lognormally distributed.

For the probability bounds analysis, the Land statistic was used to determine the lower and upper 95% confidence limits on the mean. The resulting values were used to parameterize a lognormal distribution for each fish prey group.

*Middle Calcasieu River Area of Concern (MCR AOC)*

A number of group 1, 2a and 2b fish species including gulf killifish, gulf menhaden and spot, group 3a and 3b fish species including striped mullet and spotted seatrout, and group 4a and 4b fish species including black drum and gizzard shad were sampled in the MCR AOC of the Calcasieu Estuary. These were analyzed for total mercury concentrations in fish. The fitted mean for group 1, 2a and 2b fish was 0.0589 mg/kg ww with a standard deviation of 0.0257. The fitted mean for 3a and 3b fish was 0.0525 mg/kg ww with a standard deviation of 0.0245 and group 4a and 4b fish had a fitted mean of 0.0876 mg/kg ww with a standard deviation of 0.0699. During long exposures, piscivorous mammals will spatially and temporally average their exposures. To represent this averaging, we used a bootstrapping process to sample from the mercury in fish distribution over a period of 160 days. Thus, piscivorous mammals were assumed to forage over 160 days, each day consuming group 1, 2a and 2b fish having a lognormal distribution with a mean of 0.0589 mg/kg ww and standard deviation of 0.0257, group 3a and 3b fish having a lognormal distribution with a mean of 0.0525 mg/kg ww and standard deviation of 0.0245, and group 4a and 4b fish having a lognormal distribution with a mean of 0.0876 mg/kg ww and standard deviation of 0.0699. The resulting grand mean and grand standard deviation for the 160 days was 0.0589 mg/kg ww and 0.000791 for group 1, 2a and 2b fish, 0.0524 mg/kg ww and 0.000689 for group 3a and 3b fish, and 0.0873 mg/kg ww and 0.00235 for group 4a and 4b fish, respectively. This variable was assumed to be lognormally distributed.

For the probability bounds analysis, the Land statistic was used to determine the lower and upper 95% confidence limits on the mean. The resulting values were used to parameterize a lognormal distribution for each fish prey group.

### *Reference Areas*

A number of group 1, 2a and 2b fish species including gulf killifish, gulf menhaden and spot, group 3a and 3b fish species including striped mullet and spotted seatrout, and group 4a and 4b fish species including black drum and gizzard shad were sampled in the reference areas of the Calcasieu Estuary. These were analyzed for total mercury concentrations in fish. The fitted mean for group 1, 2a and 2b fish was 0.0244 mg/kg ww with a standard deviation of 0.00970. The fitted mean for 3a and 3b fish was 0.0267 mg/kg ww with a standard deviation of 0.0161 and group 4a and 4b fish had a fitted mean of 0.0696 mg/kg ww with a standard deviation of 0.0954. During long exposures, piscivorous mammals will spatially and temporally average their exposures. To represent this averaging, we used a bootstrapping process to sample from the mercury in fish distribution over a period of 160 days. Thus, piscivorous mammals were assumed to forage over 160 days, each day consuming group 1, 2a and 2b fish having a lognormal distribution with a mean of 0.0244 mg/kg ww and standard deviation of 0.00970, group 3a and 3b fish having a lognormal distribution with a mean of 0.0267 mg/kg ww and standard deviation of 0.0161, and group 4a and 4b fish having a lognormal distribution with a mean of 0.0696 mg/kg ww and standard deviation of 0.0954. The resulting grand mean and grand standard deviation for the 160 days was 0.0244 mg/kg ww and 0.000255 for group 1, 2a and 2b fish, 0.0268 mg/kg ww and 0.000510 for group 3a and 3b fish, and 0.0698 mg/kg ww and 0.00291 for group 4a and 4b fish, respectively. This variable was assumed to be lognormally distributed.

For the probability bounds analysis, the Land statistic was used to determine the lower and upper 95% confidence limits on the mean. The resulting values were used to parameterize a lognormal distribution for each fish prey group.

### ***Concentration of Methylmercury in Invertebrates (C<sub>i</sub>)***

Concentrations of methylmercury were available for invertebrate groups 1a and 1b (*Rangia* and fiddler crab). For group 2a (shrimp), total mercury concentrations were used as a surrogate for methylmercury, when methylmercury concentrations were not available. An analysis of samples having both measured concentrations of methylmercury and total mercury showed that methylmercury comprised a large part of total mercury for 2a invertebrates. In 87% of these samples, methylmercury concentrations were within 20% of total mercury concentrations. Some of the discrepancy might be due to experimental error.

### ***Bayou d'Inde Area of Concern (BI AOC)***

A number of group 1a, 1b and 2a invertebrate animals were sampled in the BI AOC of the Calcasieu Estuary, primarily shrimp. The fitted mean was 0.0373 mg/kg ww with a standard deviation of 0.0123. The grand mean for the 160 days calculated using the bootstrapping technique was 0.0374 mg/kg ww and the standard deviation was 0.000357. This variable was assumed to be lognormally distributed.

For the probability bounds analysis, the Land statistic was used to determine the lower and upper 95% confidence limits on the mean. The resulting values were used to parameterize a lognormal distribution.

### ***Upper Calcasieu River Area of Concern (UCR AOC)***

A number of group 1a, 1b and 2a invertebrate animals were sampled in the UCR AOC of the Calcasieu Estuary, primarily shrimp. The fitted mean was 0.0198 mg/kg ww with a standard deviation of 0.0119. The grand mean for the 160 days calculated using the bootstrapping technique was 0.0197 mg/kg ww and the standard deviation was 0.000408. This variable was assumed to be lognormally distributed.

For the probability bounds analysis, the Land statistic was used to determine the lower and upper 95% confidence limits on the mean. The resulting values were used to parameterize a lognormal distribution.

*Middle Calcasieu River Area of Concern (MCR AOC)*

A number of group 2a invertebrate animals were sampled in the MCR AOC of the Calcasieu Estuary, primarily shrimp. The fitted mean was 0.0242 mg/kg ww with a standard deviation of 0.00437. The grand mean for the 160 days calculated using the bootstrapping technique was 0.0241 mg/kg ww and the standard deviation was 0.000153. This variable was assumed to be lognormally distributed.

For the probability bounds analysis, the Land statistic was used to determine the lower and upper 95% confidence limits on the mean. The resulting values were used to parameterize a lognormal distribution.

*Reference Areas*

A number of group 2a invertebrate animals were sampled in the reference areas of the Calcasieu Estuary, primarily shrimp. The fitted mean was 0.00751 mg/kg ww with a standard deviation of 0.00174. The grand mean for the 160 days calculated using the bootstrapping technique was 0.00750 mg/kg ww and the standard deviation was 0.0000536. This variable was assumed to be lognormally distributed.

For the probability bounds analysis, the Land statistic was used to determine the lower and upper 95% confidence limits on the mean. The resulting values were used to parameterize a lognormal distribution.

### ***Concentration of TCDD-TEQs in Fish***

#### ***Bayou d'Inde Area of Concern (BI AOC)***

A number of group 1, 2a and 2b fish species including gulf killifish, gulf menhaden and spot, group 3a and 3b fish species including striped mullet and spotted seatrout, and group 4a and 4b fish species including black drum and gizzard shad were sampled in the BI AOC of the Calcasieu Estuary. These were analyzed for TCDD-TEQs concentrations in fish. The fitted mean for group 1, 2a and 2b fish was 29.6 ng/kg ww with a standard deviation of 14.4. The fitted mean for 3a and 3b fish was 55.2 ng/kg ww with a standard deviation of 70.4 and group 4a and 4b fish had a fitted mean of 87.6 ng/kg ww with a standard deviation of 223. Using the bootstrapping procedure, the grand mean and grand standard deviation for the 160 days was 29.6 ng/kg ww and 0.141 for group 1, 2a and 2b fish, 54.5 ng/kg ww and 1.90 for group 3a and 3b fish, and 87.9 ng/kg ww and 6.68 for group 4a and 4b fish, respectively. This variable was assumed to be lognormally distributed.

For the probability bounds analysis, the Land statistic was used to determine the lower and upper 95% confidence limits on the mean. The resulting values were used to parameterize a lognormal distribution for each of the fish prey groups.

#### ***Upper Calcasieu River Area of Concern (UCR AOC)***

A number of group 1, 2a and 2b fish species including gulf killifish, gulf menhaden and spot, group 3a and 3b fish species including striped mullet and spotted seatrout, and group 4a and 4b fish species including black drum and gizzard shad were sampled in the UCR AOC of the Calcasieu Estuary. These were analyzed for TCDD-TEQs concentrations in fish. The fitted mean for group 1, 2a and 2b fish was 7.52 ng/kg ww with a standard deviation of 7.03. The fitted mean for 3a and 3b fish was 30.1 ng/kg ww with a standard deviation of 31.3 and group 4a and 4b fish had a fitted mean of 26.0 ng/kg ww with a standard deviation of 50.4. Using the bootstrapping

procedure, the grand mean and grand standard deviation for the 160 days was 7.49 ng/kg ww and 0.215 for group 1, 2a and 2b fish, 29.9 ng/kg ww and 0.884 for group 3a and 3b fish, and 25.5 ng/kg ww and 1.36 for group 4a and 4b fish, respectively. This variable was assumed to be lognormally distributed.

For the probability bounds analysis, the Land statistic was used to determine the lower and upper 95% confidence limits on the mean. The resulting values were used to parameterize a lognormal distribution for each fish prey group.

*Middle Calcasieu River Area of Concern (MCR AOC)*

A number of group 1, 2a and 2b fish species including sheepshead minnow, anchovy, and spot, group 3a and 3b fish species including red drum and spot, and group 4a and 4b fish species including red drum and gizzard shad were sampled in the MCR AOC of the Calcasieu Estuary. These were analyzed for TCDD-TEQs concentrations in fish. The fitted mean for group 1, 2a and 2b fish was 16.3 ng/kg ww with a standard deviation of 14.8. The fitted mean for 3a and 3b fish was 14.3 ng/kg ww with a standard deviation of 16.9 and group 4a and 4b fish had a fitted mean of 31.9 ng/kg ww with a standard deviation of 64.2. Using the bootstrapping procedure, the grand mean and grand standard deviation for the 160 days was 16.3 ng/kg ww and 0.482 for group 1, 2a and 2b fish, 14.3 ng/kg ww and 0.561 for group 3a and 3b fish, and 31.1 ng/kg ww and 1.68 for group 4a and 4b fish, respectively. This variable was assumed to be lognormally distributed.

For the probability bounds analysis, the Land statistic was used to determine the lower and upper 95% confidence limits on the mean. The resulting values were used to parameterize a lognormal distribution for each fish prey group.

### *Reference Areas*

A number of group 1, 2a and 2b fish species including gulf killifish, gulf menhaden and spot, group 3a and 3b fish species including striped mullet and spotted seatrout, and group 4a and 4b fish species including black drum and gizzard shad were sampled in the reference areas of the Calcasieu Estuary. These were analyzed for TCDD-TEQs concentrations in fish. The fitted mean for group 1, 2a and 2b fish was 7.64 ng/kg ww with a standard deviation of 2.89. The fitted mean for 3a and 3b fish was 21.4 ng/kg ww with a standard deviation of 0 and group 4a and 4b fish had a fitted mean of 3.92 ng/kg ww with a standard deviation of 2.94. Using the bootstrapping procedure, the grand mean and grand standard deviation for the 160 days was 7.64 ng/kg ww and 0.0934 for group 1, 2a and 2b fish, 21.4 ng/kg ww and 0 for group 3a and 3b fish, and 3.90 ng/kg ww and 0.0862 for group 4a and 4b fish, respectively. This variable was assumed to be lognormally distributed.

For the probability bounds analysis, the Land statistic was used to determine the lower and upper 95% confidence limits on the mean. The resulting values were used to parameterize a lognormal distribution for each fish prey group.

### ***Concentration of TCDD-TEQs in Invertebrates***

#### *Bayou d'Inde Area of Concern (BI AOC)*

A number of group 2a invertebrate animals were sampled in the BI AOC of the Calcasieu Estuary, primarily shrimp. These were analyzed for TCDD-TEQs concentrations in aquatic invertebrates. The fitted mean was 22.3 ng/kg ww with a standard deviation of 14.4. The grand mean for the 160 days calculated using the bootstrapping technique was 22.3 ng/kg ww and the standard deviation was 0.462. This variable was assumed to be lognormally distributed.

For the probability bounds analysis, the Land statistic was used to determine the lower and upper 95% confidence limits on the mean. The resulting values were used to parameterize a lognormal distribution.

*Upper Calcasieu River Area of Concern (UCR AOC)*

A number of group 2a invertebrate animals were sampled in the UCR AOC of the Calcasieu Estuary, primarily shrimp. These were analyzed for TCDD and equivalents concentrations in aquatic invertebrates. The fitted mean was 5.27 ng/kg ww with a standard deviation of 2.67. The grand mean for the 160 days calculated using the bootstrapping technique was 5.26 ng/kg ww and the standard deviation was 0.0834. This variable was assumed to be lognormally distributed.

For the probability bounds analysis, the Land statistic was used to determine the lower and upper 95% confidence limits on the mean. The resulting values were used to parameterize a lognormal distribution.

*Middle Calcasieu River Area of Concern (MCR AOC)*

A number of group 2a invertebrate animals were sampled in the MCR AOC of the Calcasieu Estuary, primarily shrimp. These were analyzed for TCDD and equivalents concentrations in aquatic invertebrates. The fitted mean was 7.09 ng/kg ww with a standard deviation of 0.603. The grand mean for the 160 days calculated using the bootstrapping technique was 7.09 ng/kg ww and the standard deviation was 0.0179. This variable was assumed to be lognormally distributed.

For the probability bounds analysis, the Land statistic was used to determine the lower and upper 95% confidence limits on the mean. The resulting values were used to parameterize a lognormal distribution.

### *Reference Areas*

No data were available.

### ***Concentration of Selenium in Fish***

#### *Bayou d'Inde Area of Concern (BI AOC)*

A number of group 1, 2a and 2b fish species including gulf killifish, gulf menhaden and spot, group 3a and 3b fish species including striped mullet and spotted seatrout, and group 4a and 4b fish species including black drum and gizzard shad were sampled in the BI AOC of the Calcasieu Estuary. These were analyzed for selenium concentrations in fish. The fitted mean for group 1, 2a and 2b fish was 0.563 mg/kg ww with a standard deviation of 0.198. The fitted mean for 3a and 3b fish was 0.526 mg/kg ww with a standard deviation of 0.152 and group 4a and 4b fish had a fitted mean of 0.480 mg/kg ww with a standard deviation of 0.140. Using the bootstrapping procedure, the grand mean and grand standard deviation for the 160 days was 0.563 mg/kg ww and 0.00612 for group 1, 2a and 2b fish, 0.526 mg/kg ww and 0.00482 for group 3a and 3b fish, and 0.480 mg/kg ww and 0.00434 for group 4a and 4b fish, respectively. This variable was assumed to be lognormally distributed.

For the probability bounds analysis, the Land statistic was used to determine the lower and upper 95% confidence limits on the mean. The resulting values were used to parameterize a lognormal distribution for each fish prey group.

#### *Middle Calcasieu River Area of Concern (MCR AOC)*

A number of group 1, 2a and 2b fish species including gulf killifish, gulf menhaden and spot, group 3a and 3b fish species including striped mullet and spotted seatrout, and group 4a and 4b fish species including black drum and gizzard shad were sampled in the MCR AOC of the Calcasieu Estuary. These were analyzed for selenium concentrations in fish. The fitted mean for group 1, 2a and 2b fish was 0.502 mg/kg

ww with a standard deviation of 0.482. The fitted mean for 3a and 3b fish was 0.758 mg/kg ww with a standard deviation of 0.645 and group 4a and 4b fish had a fitted mean of 0.698 mg/kg ww with a standard deviation of 0.203. Using the bootstrapping procedure, the grand mean and grand standard deviation for the 160 days was 0.498 mg/kg ww and 0.0146 for group 1, 2a and 2b fish, 0.759 mg/kg ww and 0.0204 for group 3a and 3b fish, and 0.697 mg/kg ww and 0.00612 for group 4a and 4b fish, respectively. This variable was assumed to be lognormally distributed.

For the probability bounds analysis, the Land statistic was used to determine the lower and upper 95% confidence limits on the mean. The resulting values were used to parameterize a lognormal distribution for each fish prey group.

#### *Reference Areas*

A number of group 1, 2a and 2b fish species including gulf killifish, gulf menhaden and spot, group 3a and 3b fish species including striped mullet and spotted seatrout, and group 4a and 4b fish species including black drum and gizzard shad were sampled in the reference areas of the Calcasieu Estuary. These were analyzed for selenium concentrations in fish. The fitted mean for group 1, 2a and 2b fish was 0.305 mg/kg ww with a standard deviation of 0.180. The fitted mean for 3a and 3b fish was 0.680 mg/kg ww with a standard deviation of 0.448 and group 4a and 4b fish had a fitted mean of 0.463 mg/kg ww with a standard deviation of 0.239. Using the bootstrapping procedure, the grand mean and grand standard deviation for the 160 days was 0.304 mg/kg ww and 0.00543 for group 1, 2a and 2b fish, 0.678 mg/kg ww and 0.0136 for group 3a and 3b fish, and 0.464 mg/kg ww and 0.00773 for group 4a and 4b fish, respectively. This variable was assumed to be lognormally distributed.

For the probability bounds analysis, the Land statistic was used to determine the lower and upper 95% confidence limits on the mean. The resulting values were used to parameterize a lognormal distribution for each fish prey group.

### ***Concentration of Selenium in Invertebrates***

#### ***Bayou d'Inde Area of Concern (BI AOC)***

A number of group 1a, 1b and 2a invertebrate animals were sampled in the BI AOC of the Calcasieu Estuary, primarily shrimp. These were analyzed for selenium concentrations in aquatic invertebrates. The fitted mean was 0.460 mg/kg ww with a standard deviation of 0.0434. The grand mean for the 160 days calculated using the bootstrapping technique was 0.457 mg/kg ww and the standard deviation was 0.00128. This variable was assumed to be lognormally distributed.

For the probability bounds analysis, the Land statistic was used to determine the lower and upper 95% confidence limits on the mean. The resulting values were used to parameterize a lognormal distribution.

#### ***Middle Calcasieu River Area of Concern (MCR AOC)***

A number of group 2a invertebrate animals were sampled in the MCR AOC of the Calcasieu Estuary, primarily shrimp. These were analyzed for selenium concentrations in aquatic invertebrates. The fitted mean was 0.464 mg/kg ww with a standard deviation of 0.175. The grand mean for the 160 days calculated using the bootstrapping technique was 0.463 mg/kg ww and the standard deviation was 0.00533. This variable was assumed to be lognormally distributed.

For the probability bounds analysis, the Land statistic was used to determine the lower and upper 95% confidence limits on the mean. The resulting values were used to parameterize a lognormal distribution.

### *Reference Areas*

A number of group 2a invertebrate animals were sampled in the reference areas of the Calcasieu Estuary, primarily shrimp. These were analyzed for selenium concentrations in aquatic invertebrates. The fitted mean was 0.424 mg/kg ww with a standard deviation of 0.186. The grand mean for the 160 days calculated using the bootstrapping technique was 0.422 mg/kg ww and the standard deviation was 0.00582. This variable was assumed to be lognormally distributed.

For the probability bounds analysis, the Land statistic was used to determine the lower and upper 95% confidence limits on the mean. The resulting values were used to parameterize a lognormal distribution.

### ***Concentration of Total PCBs in Fish***

#### *Bayou d'Inde Area of Concern (BI AOC)*

A number of group 1, 2a and 2b fish species including gulf killifish, gulf menhaden and spot, group 3a and 3b fish species including striped mullet and spotted seatrout, and group 4a and 4b fish species including black drum and gizzard shad were sampled in the BI AOC of the Calcasieu Estuary. These were analyzed for total PCBs concentrations in fish. The fitted mean for group 1, 2a and 2b fish was 0.114 mg/kg ww with a standard deviation of 0.172. The fitted mean for 3a and 3b fish was 0.364 mg/kg ww with a standard deviation of 0.879 and group 4a and 4b fish had a fitted mean of 0.602 mg/kg ww with a standard deviation of 3.078. Using the bootstrapping procedure, the grand mean and grand standard deviation for the 160 days was 0.113 mg/kg ww and 0.00513 for group 1, 2a and 2b fish, 0.362 mg/kg ww and 0.0248 for group 3a and 3b fish, and 0.602 mg/kg ww and 0.0737 for group 4a and 4b fish, respectively. This variable was assumed to be lognormally distributed.

For the probability bounds analysis, the Land statistic was used to determine the lower and upper 95% confidence limits on the mean. The resulting values were used to parameterize a lognormal distribution for each fish prey group.

### *Reference Areas*

A number of group 1, 2a and 2b fish species including gulf killifish, gulf menhaden and spot, group 3a and 3b fish species including striped mullet and spotted seatrout, and group 4a and 4b fish species including black drum and gizzard shad were sampled in the reference areas of the Calcasieu Estuary. These were analyzed for total PCBs concentrations in fish. The fitted mean for group 1, 2a and 2b fish was 0.0336 mg/kg ww with a standard deviation of 0.0580. The fitted mean for 3a and 3b fish was 0.0294 mg/kg ww with a standard deviation of 0.0350 and group 4a and 4b fish had a fitted mean of 0.0314 mg/kg ww with a standard deviation of 0.0460. Using the bootstrapping procedure, the grand mean and grand standard deviation for the 160 days was 0.0336 mg/kg ww and 0.00156 for group 1, 2a and 2b, 0.0290 mg/kg ww and 0.00105 for group 3a and 3b, and 0.0315 mg/kg ww and 0.00153 for group 4a and 4b, respectively. This variable was assumed to be lognormally distributed.

For the probability bounds analysis, the Land statistic was used to determine the lower and upper 95% confidence limits on the mean. The resulting values were used to parameterize a lognormal distribution for each fish prey group.

### ***Concentration of PCBs in Invertebrates***

#### *Bayou d'Inde Area of Concern (BI AOC)*

A number of group 1a, 1b and 2a invertebrate animals were sampled in the BI AOC of the Calcasieu Estuary, primarily shrimp. These were analyzed for total PCBs concentrations in aquatic invertebrates. The fitted mean was 0.0329 mg/kg ww with a standard deviation of 0.0264. The grand mean for the 160 days calculated using the

bootstrapping technique was 0.0354 mg/kg ww and the standard deviation was 0.000969. This variable was assumed to be lognormally distributed.

For the probability bounds analysis, the Land statistic was used to determine the lower and upper 95% confidence limits on the mean. The resulting values were used to parameterize a lognormal distribution.

#### *Reference Areas*

A number of group 2a invertebrate animals were sampled in the reference areas of the Calcasieu Estuary, primarily shrimp. These were analyzed for total PCBs concentrations in aquatic invertebrates. The fitted mean was 0.0101 mg/kg ww with a standard deviation of 0.0100. The grand mean for the 160 days calculated using the bootstrapping technique was 0.0104 mg/kg ww and the standard deviation was 0.000281. This variable was assumed to be lognormally distributed.

For the probability bounds analysis, the Land statistic was used to determine the lower and upper 95% confidence limits on the mean. The resulting values were used to parameterize a lognormal distribution.

### **2.2.1.3 Monte Carlo Analyses**

The Monte Carlo analyses for exposure combined the input distributions as per Equation 1 described in Section 2.3.1. The input distributions are summarized in Table I2-2. Each analysis included 10,000 trials and Latin Hypercube Sampling to ensure adequate sampling from all portions of the input distributions. The analyses were done in Crystal Ball 2000 (Decisioneering 2000).

#### **2.2.1.4 Probability Bounds Analyses**

The probability bounds analyses were run using Risk Calc, version 4.0 (Ferson 2002). For the probability bounds analyses, we used the input distributions used for the Monte Carlo analyses, with one exception. In the case of concentrations of contaminants in food items, the Land statistic was used to determine the lower and upper 95% confidence limits on the mean to account for uncertainty arising due to small sample sizes. The resulting values were used to parameterize lognormal distributions. When the coefficient of variation was greater than one, the minimum and maximum concentrations of the samples were taken to express uncertainty about the mean.

#### **2.2.2 Effects Assessment**

The purpose of this section is to: (1) briefly review the literature on the effects of dietary methylmercury (MeHg), TCDD-TEQs, selenium and PCBs to piscivorous mammals; and, (2) select the appropriate effects metric for each COC to use with the results of the exposure assessment to estimate risk. We will focus on ecologically relevant effects endpoints such as survival, reproduction and growth. Examples of piscivorous mammals species considered in this section include river otter (*Lutra canadensis*), and mink (*Mustela vison*). Because the available toxicological information for these species is limited for some COCs, data from other mammal studies will be discussed where appropriate. Other information on the toxicity of methylmercury, TCDD-TEQs, selenium and PCBs to wildlife can be found in the problem formulation document (MacDonald *et al.* 2001).

Effects data can be characterized and summarized in a variety of ways ranging from benchmarks designed to be protective of most or all species to dose-response curves

for the receptor group of interest (i.e., piscivorous mammals). In this assessment, effects characterization will preferentially rely on dose-response curves, but may default to benchmarks or other estimates of effect [e.g., no observed adverse effect level (NOAEL), lowest observed adverse effect level (LOAEL)] when insufficient data are available to derive dose-response curves. Effects associated with growth, survival, and reproduction are generally the preferred measures of effect. The objective of this section is to provide an overview of the available options for characterizing effects information and to describe the decision criteria for choosing among them for piscivorous mammals exposed to the COCs.

The following is the hierarchy of decision criteria used to characterize effects for each COC:

1. Had bioassays with five or more treatments been conducted on the receptor group of interest or a reasonable surrogate? If yes, we estimated the dose-response relationship using the Generalized Linear Model (GLiM) framework described in Kerr and Meador (1996) and Bailer and Oris (1997). The GLiM framework involves conducting linear regression analysis on dose-response data that have been transformed to linearize the relationship (e.g., probit transformation for survival data). If not, we proceeded to 2.
2. Were multiple bioassays available that, when combined, had five or more treatments on the receptor group of interest or a reasonable surrogate? Such bioassays would be expected to have had similar protocols, exposure scenarios and effects metrics. If yes, we estimated the dose-response relationship as in 1. If not, we proceeded to 3.

3. Had bioassays with less than five treatments been conducted on the receptor group of interest or a reasonable surrogate? If yes, we conducted hypothesis testing to determine the NOAEL and LOAEL or reported these metrics when available from the original study. If not, we proceeded to 4.
4. Were sufficient data available from field studies and monitoring programs to estimate concentrations or doses of COCs consistently associated with no adverse effects and with adverse effects to piscivorous mammals? If yes, we developed field-based no effects and effects measures. This approach is analogous to the approach used to develop sediment-quality guidelines for the protection of aquatic life (see Long *et al.* 1995; MacDonald *et al.* 1996; MacDonald *et al.* 2000b). If not, we proceeded to 5.
5. We derived a range within which the threshold for the receptor group of interest was expected to occur. Because information on the sensitivity of the receptor of interest was lacking, it was difficult to derive a threshold that was neither biased high or low. If bioassay data were available for several other species, however, one could calculate a threshold for each to determine a threshold range that spanned sensitive and tolerant species. That range was assumed to include the threshold for the receptor group of interest.

### **2.2.2.1 Mercury**

Mercury has no known physiological use to mammals, but has teratogenic, mutagenic, and carcinogenic effects (Eisler 2000). Mercury most commonly exists as methylmercury (MeHg) in higher trophic level species (Wolfe *et al.* 1998). MeHg attacks the central nervous system, affecting coordination, sight, hearing, and sensory

functions (Eisler 2000). Acute effects of MeHg include muscular uncoordination, falling, slowness, calmness, and hyperactivity (USEPA 1997b). Chronic exposure may lead to liver or kidney damage, neurobehavioral effects, reduced food consumption, weight loss, impaired growth, effects to reproduction and growth (USEPA 1997b).

### ***Survival***

Mink fed a dietary concentration of 0.58 mg/kg MeHg exhibited no obvious signs of mercury (Hg) poisoning after 25, 50, 75, or 100 d of exposure (Jernelöv *et al.* 1976). In a two generation study on mink, Dansereau *et al.* (1999) fed diets of 0.1 and 0.5 mg/kg total (T) Hg which had no effect on survival after a 704 day exposure. However, a concentration of 1.0 mg/kg T Hg killed 30 of 50 first generation mink after 90 days of exposure. In the second generation, 6 of 7 mink in the 1.0 mg/kg T Hg died after 330 d of exposure. Wren *et al.* (1987a) also found similar effects in male and female mink fed 1.0 mg/kg MeHg for 180 d. Eight of 12 females and 1 of 4 males died during the experiment. Chamberland *et al.* (1996) conducted chronic oral studies on mink fed daily diets of 0.1, 0.5 and 0.9 mg/kg of Hg for 109 days. The diets were composed of fish that were naturally contaminated with methylmercury. The study consisted of 20 mink per group of which 1, 0 and 5 died in the 0.1, 0.5 and 0.9 mg/kg groups of total mercury, respectively.

The onset of adverse effects occurs more quickly as the dietary concentration increases. All mink exposed to dietary levels of 1.1, 1.8, 4.8, 8.3, or 15 mg/kg MeHgCl showed histopathological effects after 93 d of exposure (Wobeser *et al.* 1976). Mink in the 1.8-15 mg/kg exposure groups developed clinical signs of Hg poisoning within the experimental period. Clinical signs included anorexia, weight loss, head tremors, ataxia, and convulsions; 100% mortality occurred in all of these groups.

Aulerich *et al.* (1974) fed mink 5 mg/kg MeHg. Effects to mink were similar to those in the 4.8 mg/kg MeHgCl dietary concentration group of Wobeser *et al.* (1976). Clinical signs including anorexia, uncoordination, and convulsions were apparent after a latency period of 24-d. Death usually occurred after 33-d of exposure (Aulerich *et al.* 1974).

River otters display effects similar to those reported in mink. O'Connor and Nielsen (1981) fed river otters either 2, 4, or 8 mg/kg MeHg. The average survival times for each of the treatment groups was 54-, 117-, and 184-d. The onset of intoxication correlated with the dietary concentration and all exposed river otters went through the same toxicological steps. As a result of their findings, O'Connor and Nielsen (1981) believe that adverse sublethal effects to behavior and reproduction could result from prolonged exposure to less than 2 mg/kg MeHg in the diet.

Three studies have documented adverse effects in cats (*Felis catus*) fed a daily dose of 0.25 mg/kg MeHg (Charbonneau *et al.* 1974; Eaton *et al.* 1980; Khera *et al.* 1974 in Khera 1979). Charbonneau *et al.* (1974) orally dosed 8 cats with gelatin capsules containing methylmercuric chloride dissolved in corn oil. A second group of 4 cats was fed a diet with contaminated fish. The mean survival time was 78 d and symptoms of intoxication were similar to those reported by Charbonneau *et al.* (1974; 1976).

The studies discussed for mink, river otters, and other species reported similar effects and related dietary concentrations. These studies show that MeHg can be lethal to mink at dietary levels of 1-2 mg/kg (Dansereau *et al.* 1999; Wobeser *et al.* 1976). Methylmercury is lethal to river otters at a dietary concentration of 2.0 mg/kg (O'Connor and Nielsen 1981).

### ***Reproduction***

Methylmercury can have adverse effects to young at levels considered harmless to adults (Eisler 2000). All forms of Hg can cross the placenta, but MeHg specifically concentrates in the fetal brain. Reproductive effects resulting from exposure to MeHg include developmental alterations leading to behavioral impairments after birth, as well as decreased fertility and increased occurrence of fetal death (Eisler 2000). Few studies were found on the effects of MeHg to the reproduction of mink and river otters.

Adult dogs and cats exposed to 0.1-0.25 mg/kg of MeHg chloride in the diet during pregnancy exhibited a variety of reproductive effects, such as increases in abortion, stillbirths, and irregular fetuses (Earl *et al.* 1973; Khera 1973). High incidence of stillbirths was also recorded in sows fed up to 0.5 mg/kg MeHg during pregnancy (Earl *et al.* 1973).

Wren *et al.* (1987b) observed no effect on male fertility, percentage of females whelped, or number of kits born from adult mink fed a diet containing 1.0 mg/g MeHg. Dansereau *et al.* (1999) similarly found that although the percentage of females giving birth was significantly different between dietary concentration groups, other factors like gestation period and litter size were not significantly different.

Placental transfer of Hg was demonstrated in Wren *et al.* (1987a). Mink kits born from adults fed 1.0 mg/g MeHg contained high levels of Hg on the day of whelp. At 5 weeks of age these levels had decreased, suggesting that Hg transfer through mothers milk is not a significant route of transport.

### ***Field Studies***

Field studies have shown the correlation between Hg concentration in prey and predators species. They also illustrate local and regional variation of Hg exposure. This is especially true for the transfer and biomagnification of Hg from aquatic systems to piscivorous wildlife (Wren *et al.* 1986; Sheffy and St. Amant 1982). The field studies discussed below focus on clinical symptoms of poisoning, pathological and/or histological lesions found, and the tissue concentrations in collected animals.

Sheffy and St. Amant (1982) analyzed a variety of small mammals trapped in Wisconsin between 1972-75. Their study found that otters had the highest Hg burdens followed by mink > raccoon > fox > muskrat > beaver. Mean Hg tissue concentrations for mink were: kidney 2.33; liver 2.08; and, brain 0.46 mg/kg. The maximum Hg tissue concentrations in 39 mink collected were: fur 41.2; kidney 12.5; and, liver 17.4 mg/kg. Sheffy and St. Amant (1982) found Hg tissue concentrations were higher from industrialized sections of the Wisconsin River than non-industrialized sections. Osowski *et al.* (1995) found a similar discrepancy in tissue concentrations. Declining mink populations from coastal areas of Georgia and North Carolina had mean Hg concentrations in their kidneys of 2.24 mg/kg. In contrast, mink from the Piedmont regions had mean Hg concentrations of 0.53 mg/kg.

O'Connor and Nielsen (1981) examined mink harvested by trappers in the northeastern US. Histological and pathological examinations found lesions in some of the mink. Forty-four percent of the mink examined also had lesions in their CNS. Mean liver Hg concentration was higher in males (1.20 mg/kg) than females (0.73 mg/kg; O'Connor and Nielsen 1981).

Foley *et al.* (1988) reported tissue concentrations for mink from across New York State. Mercury concentrations in liver ranged from 0.25 to 7.66 mg/kg. Wren *et al.*

(1986) discovered changes in concentration relative to the proximity of a contaminated site. Mean liver Hg concentrations in a contaminated area were 3.75 mg/kg versus 1.13 mg/kg in samples 2 km away. Total Hg concentrations did not differ significantly between males and females. A pattern was found between Hg concentrations in mink and their prey between different locations (Wren *et al.* 1986). Field studies of river otters also found tissue concentration changes with proximity to contaminated sites (Foley *et al.* 1988; Wren *et al.* 1986) and a correlation between levels in prey species and predators (Wren *et al.* 1986). Wren *et al.* (1986) sampled river otters from five study areas in Ontario. Mean liver Hg concentrations from a contaminated and an uncontaminated area were 4.57 mg/kg and 1.3 mg/kg, respectively. The highest liver Hg concentrations were 14.3 and 17.4 mg/kg. The river otter with the former concentration also had a brain Hg concentration of 7.1 mg/kg. Wren *et al.* (1986) found tissue concentration changes in prey from contaminated to uncontaminated sites. They did not find differences in Hg levels between male and female river otters within each area.

Sheffy and St. Amant's (1982) study of furbearers in Wisconsin recorded the following mean Hg concentrations for river otters: kidney 8.47; liver 3.34; and, brain 0.74 mg/kg. Maximum Hg concentrations from 41 river otters were: fur 63.2; kidney 20.9; and, liver 23.6 mg/kg. O'Connor and Nielsen (1981) reported a lower mean liver Hg concentration in river otters from the northeastern US. They also found males had a higher mean liver Hg concentration (males 2.14, females 1.12 mg/kg). Approximately half of the river otters examined had lesions in either the lungs, intestines, or bladder (O'Connor and Nielsen 1981).

In summary, field studies confirm that Hg concentrations vary with location and proximity to contaminated sites. Studies differ on Hg levels between sexes. O'Connor and Nielsen (1981) found males had higher concentrations while Wren *et*

*al.* (1986) found no difference. Although mercury-exposed animals may appear healthy and show no clinical signs of Hg toxicity, lesions to tissues and the CNS may exist.

### ***Effects Metrics***

Ideally, to derive the effects metric for piscivorous mammals, we would prefer to have long-term feeding studies that estimated effects of at least five dose levels on a sensitive endpoint, such as reproductive fecundity. No such study was found for methylmercury. However, several long-term feeding studies (30-704 days) with female mink have been conducted using similar protocols (Aulerich *et al.* 1974; Wobeser *et al.* 1976; Wren *et al.* 1987a; Chamberland *et al.* 1996; Dansereau *et al.* 1999). We combined these studies to produce a toxicity data set comprised of 16 treatment levels. The endpoint was female mortality that, although not ideal, seems to result in similar effects estimates as occurs with reproductive endpoints such as kit biomass or number of surviving kits (Wren *et al.* 1987b).

The concentration-response relationship for female mortality versus methylmercury concentration in the diet was estimated using the generalized linear model (GLiM) framework with a logit link function for survival data (i.e., linear regression analysis on concentration-response data that have had a logit transformation to linearize the relationship) and a binomial error distribution (Figure I2-4). Kerr and Meador (1996) and Bailer and Oris (1997) describe this framework in detail. The analysis was carried out in SAS® (SAS Institute, Cary, NC) and the 95% fiducial limits estimated. The model parameters were  $\beta_0 = 0.0211$ ,  $\beta_1 = -10.28$ ,  $se\beta_0 = 0.214$ ,  $se\beta_1 = 1.94$ , and  $corr\beta_0\beta_1 = 0.341$ . The resulting concentration-response curve for the mortality effects of mercury to mink produced an adequate model to fit the data ( $p < 0.0001$ ; F value = 3.96E12).

To convert effect concentrations to doses, the logit concentration-response model was combined with the food intake rate for captive female mink (Figure I2-5). Bleavins and Aulerich (1981) found that captive female mink have a mean food intake rate of 155 g/kg bw/day.

#### **2.2.2.2 TCDD-TEQs**

Coplanar PCDDs, PCDFs, and PCBs act by the same mode of toxic action, initiated by binding to the aryl hydrocarbon receptor protein (Bosveld *et al.* 1994). The response of organisms can range from mortality (Safe 1994; Eisler and Belisle 1996; Tillitt *et al.* 1996) to enzyme induction (Aulerich *et al.* 1985). The most toxic PCDD and PCDF congeners tend to be those chlorinated in the 2, 3, 7, and 8 positions, such as 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD), as this configuration best fits the receptor site. The toxic response to this group of contaminants is therefore related to the three-dimensional structure of the substance, including the degree of chlorination and positions of the chlorine atoms on the aromatic frame. Substances that are more structurally similar to TCDD will elicit a toxic response closer to that of TCDD. The toxicity of PCBs, PCDDs, and PCDFs may therefore be expressed in terms of 2,3,7,8-TCDD Toxic Equivalents (TEQs), as described by van den Berg *et al.* (1998), where the toxicity of the members of this chemical class are all expressed relative to TCDD for fish, birds, and mammals. This approach is described in further detail in Appendix G. We will use this approach to convert the toxicity test results for PCDD, PCDF, and PCB congeners described below.

#### ***Survival***

Hochstein *et al.* (1988) randomly separated 16 mink into four groups of four animals each and administered single oral doses of 2,3,7,8-TCDD at levels of 0, 2,500, 5,000,

and 7,500 ng TEQ/kg bw for 28 days. The mink exhibited a dose-related decrease in feed consumption and also experienced a dose-related decrease in body weight. Other effects from the higher doses included enlarged organs (relative to body weight), such as heart, brain and thyroid as well as discoloration and blotching of the liver and kidneys. Control animals and those dosed with 2,500 ng TEQ/kg bw all survived to the conclusion of the 28-day study, but those treated with 5,000 and 7,500 ng TEQ/kg bw survived, on average, to 12.3 and 9.5 days, respectively. The LD<sub>50</sub> for the 28 day single oral dose of TCDD was calculated to be 4,200 ng TEQ/kg bw.

The same group of authors (Hochstein *et al.* 1998) exposed female mink to 0, 1, 10, 100, 1000, 10,000, and 100,000 ng TEQ/kg TCDD in feed (estimated doses of 0, 0.14, 1.4, 14, 140, 1,400, and 14,000 ng TEQ/kg bw/day) for 125 days and observed a dose-dependent wasting syndrome (decrease in body weight). Mortality reached 12.5, 62.5, and 100% after 28 days of exposure to 140, 1,400, and 14,000 ng TEQ/kg bw/day, respectively. After 125 days of exposure, mortality reached 100% in the 1,400 and 14,000 ng/kg bw/day exposure groups.

Mature female mink fed doses of 0.6, 16, 53, 180 and 1,400 ng TEQ/kg TCDD (0.084, 2.24, 7.42, 25.2, and 196 ng TEQ/kg bw/day) for a maximum of 132 days exhibited 17% mortality at the highest dose level (Hochstein *et al.* 2001). Final body weights of adult female mink were inversely proportional to dietary TCDD concentration.

Newborn mink given doses (intraperitoneal injection) of 100 and 1,000 ng TEQ/kg bw of TCDD for 12 days experienced 100% mortality at the higher dose. The lower dose depressed body weight and produced 62% mortality (Aulerich *et al.* 1988). Adult mink administered a single oral dose of 2,500 ng TEQ/kg bw had significantly reduced body weights after three weeks (Hochstein *et al.* 1998).

Aulerich *et al.* (1987) conducted mink dietary exposure studies to PCBs and found that a dietary PCB169 concentration of 50,000 ng/kg (5,000 ng/kg TEQ diet) was sufficient to cause significant weight loss and mortality in adult female mink exposed over a period of 135 days. A previous study (Aulerich *et al.* 1985) showed 100% mink mortality within 60 days when fed a diet containing 500,000 ng/kg PCB169 (50,000 ng/kg diet TEQ) and 50% mortality in a span of 3 months when fed a diet containing 100,000 ng/kg PCB 169 (10,000 ng/kg diet TEQ).

Pohjanvirta *et al.* (1993) investigated the acute oral toxicity of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD), 1,2,3,7,8-pentachlorodibenzo-*p*-dioxin (PeCDD) and 1,2,3,4,7,8-hexachlorodibenzo-*p*-dioxin (HxCDD) to Long-Evans (LE) rats and Han/Wistar (H/W) rats given single doses by gavage. TCDD was the most toxic of the chemicals, producing LD<sub>50</sub>s of 9,800 and 17,700 ng/kg bw for female and male LE rats, respectively. The LD<sub>50</sub>s for the H/W rats were in excess of 7,200,000 ng/kg bw. PeCDD treatments revealed similar strain differences, with female LE rats having LD<sub>50</sub>s of 20,000-60,000 ng/kg bw and female H/W rats having LD<sub>50</sub>s over 1,620,000 ng/kg bw. HxCDD showed less of a strain-related difference in toxicity with a LD<sub>50</sub> for H/W female rats of 187,100 ng/kg bw compared to between 12,000-36,000 ng/kg bw for LE rats.

Sprague-Dawley rats were investigated by Stahl *et al.* (1992) with a similar complement of chemicals: TCDD; PeCDD; HxCDD; and, 1,2,3,4,6,7,8-heptachlorodibenzo-*p*-dioxin (HpCDD). The reported single dose LD<sub>50</sub>s for these substances were 43,000, 206,000, 88,700 and 63,250 ng TEQ/kg bw, respectively. Van Miller *et al.* (1977) maintained Sprague-Dawley rats on diets containing 1 to 1,000,000 ng/kg TCDD for 78 weeks. A daily dose can then be ascertained from the cumulative TCDD consumption of the animals and their average body weight over the course of the study. Doses were estimated to range from 0.0428 to 71,400 ng/kg

bw/day. Dosage levels above 3,400 ng/kg bw/day produced 100% mortality in the test animals within 3 weeks and 57.1 ng/kg bw/day produced 100% mortality within 31 weeks. Kociba *et al.* (1978) treated male and female Sprague Dawley rats with oral doses of TCDD of 0, 1, 10 and 100 ng/kg bw/day for two years. No significant effects were noted below 10 ng/kg bw/day but at 100 ng/kg bw/day, a cumulative increase in mortality ( $p<0.05$ ) in the latter half of the study period was observed, as well as a decrease in mean body weight ( $p<0.05$ ) from 6-24 months compared to controls.

### ***Reproduction***

Heaton *et al.* (1995) investigated the reproductive effects of dietary exposure to planar halogenated hydrocarbons (PCDDs, PCDFs, PCBs, and TEQs) for adult mink over a 182 day period. The study began prior to mating and exposure continued after the kits were whelped. In this study, carp were collected from contaminated and reference sites near Saginaw Bay, MI. Their TEQ body burden was measured and the fish incorporated into mink feed at levels of 0, 10, 20, and 40% contaminated carp. This resulted in TEQ dietary concentrations of 1.03, 19.4, 40.0, and 80.8 ng TEQ/kg feed and estimated daily doses of 0.25, 3.60, 6.80, and 10.7 ng TEQ/kg bw/day, as determined by the H4IIE bioassay. All of the treatment levels affected reproductive success of mink. The lowest dose significantly reduced kit body weight at three and six weeks to 67 and 79% of control body weights, respectively. At the highest dose level, the gestation length was reduced to 91% of controls, average litter size was reduced to 3.3 compared to 5.7 for controls, and the number of kits born alive per female was reduced from 5.0 for controls to 0.7 for mink fed the diet containing 40% Saginaw Bay carp. The NOAEL and LOAEL were calculated to be 0.27 and 4.23 ng TEQs/mink/day. Survivability of kits to six weeks was reduced from 85% in control animals to 28% and 11% in the 10% and 20% carp diets, respectively. No kits survived to 6 weeks in the 40% carp dietary treatment. In a subsequent publication

of this study, Tillitt *et al.* (1996) estimated a NOAEL ranging from 0.08-0.27 ng TEQ/kg bw/day and a LOAEL ranging from 2.24-3.44 ng TEQ/kg bw/day. They used two TEQ calculation methods to arrive at the ranges. Similarly, Brunstrom *et al.* (1991) reported a NOAEL and a LOAEL of 0.3 and 2.4 ng TEQ/kg bw/day associated with decreased fecundity.

Adult mink exposed to TCDD at concentrations of 0.6, 16, 53, 180, and 1,400 ng TEQ/kg (0.084, 2.24, 7.42, 25.2, and 196 ng TEQ/kg bw/day) for up to 132 days produced offspring that had reduced survival and a dose-dependent decrease in kit weight from birth to week three of exposure (Hochstein *et al.* 2001).

Mink were treated with doses of 0.25, 3.6, 6.8 and 10.7 ng TEQ/kg bw/day prior to and throughout the reproductive period to evaluate survival and reproductive effects (Heaton *et al.* 1995). At 0.25 ng/kg bw/day, 15% mortality was observed after 3 weeks of exposure. Sixty nine percent mortality in kits was reported at the exposure level of 3,600 ng/kg bw/day. The mortality increased to 100% at the dose of 10,700 ng/kg bw/day.

Holtzman rats exposed to TCDD at a dose of 1,000 ng TEQ/kg bw/day on day 15 of gestation experienced a 19% decrease in fetal survival ( $p < 0.05$ ) on day 21 of gestation (Mably *et al.* 1992). Similarly, Giavini *et al.* (1983) treated CRCD rats to doses of 2,000 ng TEQ/kg bw/day and observed that fetal survival at 21 days gestation decreased by 45% ( $p < 0.01$ ) compared to controls. Khera and Ruddick (1973) dosed pregnant Wistar rats at TCDD levels of 0, 125, 250, 500, 1,000, 2,000, 4,000, 8,000, 16,000 ng TEQ/kg bw/day on gestation days 6 to 15. At day 22, the animals were sacrificed and it was found that doses above 4,000 ng TEQ/kg bw/day produced 100% embryonic lethality. Huuskonen *et al.* (1994) treated Long-Evans rats to an oral dose of TCDD of 5,000 ng TEQ/kg bw/day on day 8 of gestation and observed

a significant ( $p < 0.05$ ) decrease in the number of living fetuses per litter. Over 70% of implantations were resorbed while 5% died at a later fetal stage. Similar observations were made by Sparschu *et al.* (1971) in Sprague Dawley rats fed doses of TCDD of 0, 30, 125, 500, 2,000 and 8,000 ng TEQ/kg bw/day on days 6-15 of gestation. Pregnancy was terminated on day 20 of gestation by decapitation. The number of viable fetuses decreased and the total number of resorptions increased dose dependently starting at 125 ng TEQ/kg bw/day. Maternal body weight gains decreased ( $p < 0.01$ ) at 500 ng TEQ/kg bw/day while fetal body weight decreased ( $p < 0.01$ ) at 125 ng TEQ/kg bw/day. Pre- and post-implantation loss due to TCDD treatment were observed by Giavini *et al.* (1983). CRCD rats were dosed at a TCDD level of 2,000 ng TEQ/kg bw/day for two weeks before mating experienced a significant increase ( $p < 0.05$ , 19.5%) in pre-implantation loss, while doses of 500 and 2,000 ng TEQ/kg bw/day produced significant increases in post-implantation loss of 10.2% and 30.3%, respectively. Significant fetal weight reduction was also observed at the 2,000 ng TEQ/kg bw/day dose.

The number of pups and survival rates of newborns have been reported to decrease as a result of treating dams with TCDD. Murray *et al.* (1979) treated Sprague Dawley rats with diets of 0, 1, 10 or 100 ng TEQ/kg bw/day for 90 days. The dosage period began at 7 weeks and ran for 90 days, at which time the F0 rats were mated to produce the F1a generation. The F0 rats were then mated again 33 days later to produce the F1b generation. The F1a and F1b rats were mated at an average age of 130 days to produce the F2 and F3 generations, respectively. At 100 ng TEQ/kg bw/day, the F1a and F1b generations were smaller in number ( $p < 0.05$ ) and F1a pups were all stillborn. At 10 ng TEQ/kg bw/day, the F2 and F3 generations were smaller in number ( $p < 0.05$ ) and had significantly lower survival rates to 21 days, 86% and 83%, compared to 90% for controls.

Holtzman rats receiving a single oral dose of TCDD of 1,000 ng/kg bw/day on day 15 of gestation (Bjerke and Peterson 1994; Mably *et al.* 1992) experienced a significant reduction in body weight of offspring at birth and at 5 days postpartum. Offspring survival rates were significantly reduced as well, with Bjerke and Peterson (1994) reporting a 30% decline and Mably *et al.* (1992) reporting a 15% decline.

Bjerke *et al.* (1994) dosed Holtzman rats with 700 ng TEQ/kg bw on day 15 of gestation by gavage and observed survival and body weights to 7 days. This study included pup exposure both in utero and through lactation. Pup mortality was significantly higher in test animals than in controls (23% vs 7%) and mean body weight of male offspring was decreased by 89-92% of control between birth and 7 days post partum.

Gray and Ostby (1995) administered 1,000 ng TEQ/kg bw of TCDD by gavage in Holtzman rats on gestation day 8 and observed significantly reduced fertility of female offspring compared to controls. Breeding of female offspring was monitored for 140 days starting at 233 days of age and only 19% produced a fifth litter compared to 61% for the control. Khera and Ruddick (1973) also found a decrease in the pregnancy rate and average litter size of dams administered a dose of 500 ng TEQ/kg bw/day TCDD during gestation days 6 to 15. There was no effect at 250 ng TEQ/kg bw/day.

2,3,4,7,8-pentachlorodibenzofuran was observed to cause fetal mortality in rats. Couture *et al.* (1989) treated Fisher 344 rats with a single dose of 2,3,4,7,8-pentachlorodibenzofuran ranging from 0 to 150,000 ng TEQ/kg bw/day on days 8, 10 or 12 of gestation with the animals being sacrificed on day 20. Rats dosed with 50,000 ng TEQ/kg bw on day 8 of gestation had significantly higher fetal mortality (9.93%) compared to controls (2.78%). The dose of 150,000 ng TEQ/kg bw

administered on days 10 and 12 of gestation also produced significantly higher fetal mortality (91.7% and 81.1%, respectively) compared to controls (0% and 7.86%). Fetal weight was correlated with dose and fetal toxicity, and teratogenic effects were observed at the highest dose for administration on all three days, though significantly on days 8 and 12 only.

Rats and mice treated with 3,3',4,4'-tetrachlorobiphenyl (PCB77) suffered embryotoxic effects to the fetuses including death and resorption (Marks *et al.* 1989; d'Argy *et al.* 1987; Wardell *et al.* 1982). Wardell *et al.* (1982) observed significant embryonic mortality (14% resorption) in Sprague Dawley rats when dams were exposed to a dose of 300 ng TEQ/kg bw/day of PCB77 on days 6-18 of gestation. d'Argy *et al.* (1987) reported a significant number of resorptions (37%) when C57BL/6 (B6) mice were administered an oral dose of 2,500 ng TEQ/kg bw/day of 3,3',4,4'-pentachlorobiphenyl (PCB77) on day 11 of gestation.

Marks *et al.* (1989) reported a PCB77 dose-related increase of the percentage of implants resorbed by mice administered 400 (7%) to 6,400 (82.5%) ng TEQ/kg bw/day; a significant increase was determined at 1,600 (16.4%) ng TEQ/kg bw/day. In addition, the average number of live fetuses per dam was significantly reduced (21.5%) at 1,600 ng TEQ/kg bw/day and above (Marks *et al.* 1989). Rands *et al.* (1982) observed that pregnant rats dosed with 300 ng TEQ/kg bw/day of PCB77 on day 6 to 18 of gestation experienced a statistically significant increase in mortality of offspring. The results also showed a trend toward decreased viability with increasing gestational time; this was also observed by Linzey (1987).

### ***Field Surveys***

No field surveys or studies were found in the literature involving the exposure of piscivorous mammals to TCDD-TEQs.

### ***Effects Metrics***

To derive dose-response curves, we require long-term feeding studies and reported responses at several dose levels. The responses should include relevant and sensitive endpoints such as mortality, reproduction, or growth. There were five studies that met these criteria. The studies were Heaton *et al.* (1995), Tillitt *et al.* (1996), Hochstein *et al.* (1998; 2001), and Aulerich *et al.* (1988). However, most of these studies employed field-collected fish that contained contaminants other than dioxins or dioxin-like compounds. Therefore, the observed responses could not be attributed exclusively to the contaminants of interest, and interference from other unknown contaminants could not be ruled out. The effects metrics for TCDD-TEQs are based on the combined investigations conducted by Khera and Ruddick (1973) and Sparschu *et al.* (1971). These investigators both treated pregnant female rats (Wistar and Sprague-Dawley, respectively) with oral doses of TCDD (8 and 5 treatment levels, respectively) by gavage on days 6 to 15 (inclusive) of gestation. The authors reported a number of reproductive endpoints, including the number of live fetuses per female. Generally, there were no significant effects on the number of live fetuses per female at doses under 1,000 ng/kg bw/d, but effects rose sharply to 100% mortality as the dose exceeded 4,000 ng/kg bw/day.

The dose-response relationship for fecundity versus TCDD-TEQs concentration in the diet was estimated using the generalized linear model (GLiM) framework with a log link function for number of fetuses per female data (i.e., linear regression analysis on dose-response data that have had a log transformation to linearize the relationship) and a Poisson error distribution (Figure I2-6). Kerr and Meador (1996) and Bailer and Oris (1997) describe this framework in detail. The analysis was carried out in SAS® (SAS Institute, Cary, NC). The model parameters were  $\beta_0 = 2.49$ ,  $\beta_1 = -0.677$ ,  $se\beta_0 = 0.107$ ,  $se\beta_1 = 0.147$ , and  $corr\beta_0\beta_1 = -0.505$ . The resulting dose-response curve

for fecundity of TCDD-TEQs to rat produced an adequate model fit to the data ( $p < 0.0001$ ; F value= 88.64).

The use of rat toxicity data for piscivorous mammals should be viewed with caution because rats are quite resistant to the toxic effects of dioxins compared to mink, which are considered very sensitive. Rat toxicity data will likely underestimate the level of risk to mink (Birnbaum and Toumisto 2000).

### **2.2.2.3 Selenium**

Selenium is an essential element in human and animal nutrition and is efficiently concentrated in living tissues. Absorption of oral radioselenite by rats is as high as 95 to 100% (Eisler 1985). Marine fish have tissue residues of approximately 2 mg/kg ww, a concentration 50,000 times that of the surrounding seawater (Wilber 1980). Though essential to life and naturally accumulated, excess selenium exposure has been associated with lethality, neurological, developmental, and reproductive effects (ATSDR 1996). The selenium compounds shown to be the most toxic to mammals by ingestion appear to be sodium selenite and sodium selenate (Olson 1986). Both sodium selenate and sodium selenite are used as livestock feed supplements to prevent selenium deficiency diseases and both have been detected at chemical waste sites (NTP 1994).

#### ***Survival***

Some of the earliest toxicological work done with selenium was conducted by Franke and Moxon (1936). These investigators established the median lethal intraperitoneal dose of sodium selenate to rats at 5.25 to 5.75 mg/kg bw and sodium selenite at 3.25 to 3.5 mg/kg bw. Other forms of selenium were shown to be less toxic by other

researchers: diselenodipropionic acid with a reported LD<sub>50</sub> of 25 mg/kg bw (Moxon *et al.* 1938); trimethylselenonium LD<sub>50</sub> at 49.4 mg/kg bw (Obermeyer *et al.* 1971); dimethyl selenide at LD<sub>50</sub> of 1,600 mg/kg bw (McConnell and Portman 1952); and, elemental selenium at LD<sub>50</sub> of 6,700 mg/kg bw (Cummins and Kimura 1971). Pletnikova (1970) examined the oral route of exposure and established the single oral dose LD<sub>50</sub> for sodium selenite to the white mouse to be 7.75 mg/kg bw. The albino rat was also tested and had an oral LD<sub>50</sub> of 10.5 mg/kg bw. Smith and Westfall (1937) claimed that the route of administration was not an important factor in selenium toxicity due to the rapid and complete absorption of soluble selenium compounds.

Adult female CD-1 rats were administered sodium selenite by gavage for 8 days at levels of 2.5, 5, 10, 20, and 40 mg/kg/day in a study by Plasterer *et al.* (1985). Mice were treated in groups of ten and all were 61 to 71 days old at the initiation of the experiment. There was no evidence of a weight change in the animals and mortality was observed in each of the treatment levels. The LD<sub>50</sub> was established, using probit analysis, at 8.4 mg/kg bw/day with a 95% confidence limit of 6.0 - 12.0 mg/kg bw/day. The 8 day LD<sub>10</sub> was determined to be 7.0 mg/kg bw/day.

The effects of sodium selenite were also investigated with respect to the short- and long-term survival of Sprague-Dawley rats (Jacobs and Forst 1981). Five groups of five females were provided with water *ad libitum* treated with sodium selenite at concentrations of 1, 4, 8, 16, and 64 mg/L Se. Using an estimated body weight of 204 g and daily water intake of 31 mL (TERA 2002), the drinking water concentrations translate to daily doses of 0.15, 0.61, 1.22, 2.44, and 9.73 mg/kg bw/day for young female rats. For the 35 week exposure, rats were started at 5 or 12 weeks of age and monitored for growth and survival until death or the conclusion of the experiment. The group started at 5 weeks experienced significant mortality starting at 16 mg/L and

complete mortality at the 64 mg/L treatment level. Animals that were started on the treatment at 12 weeks of age experienced significant mortality only at the highest treatment level, with all animals dying within 18 days. Growth was also measured for these two groups, with weight gain exhibiting a negative correlation with treatment level. In both groups, males and female rats both lost weight over the course of the experiment while rats in the control and lower level treatments gained weight. Longer studies involving similar animals, procedures, and measurement endpoints were conducted with exposure periods of 61 and 116 weeks to 4 mg/L selenium in drinking water (Jacobs and Forst 1981). The longer exposures elicited no adverse effects to rats for survival or reproduction.

Schroeder and Mitchener (1971a) exposed weanling Long-Evans rats to sodium selenate or sodium selenite in drinking water for 1 year at 0 or 2 mg selenium/L. After 1 year, the selenium selenate concentration was increased to 3 mg/L. The group given the sodium selenate performed as well as controls, both reaching 90% mortality at approximately 1100 days. The male rats given the sodium selenite drinking water solution reached 50% mortality in 58 days, and females in 342 days. There was also a significant lag in body weight gain in males and female rats. The drinking water concentration of 2 mg/L was estimated to translate to a daily dose of 0.28 mg Se/kg bw/day (ATSDR 1996). Rosenfeld and Beath (1954) provided drinking water containing potassium selenate to rats at a concentration such that a daily dose of 1.05 mg Se/kg bw/day was achieved. The exposure period was 8 months and no mortalities were observed.

A study commissioned by the National Toxicology Program (NTP 1994) investigated the effects of sodium selenate given to rats and mice in drinking water over 13 weeks. Animals were divided into single sex groups of 10 and given drinking water treated with levels of 0, 3.75, 7.5, 15, 30, or 60 mg/L sodium selenate. At the conclusion of

the study, the surviving animals were sacrificed and all were examined for hematology, clinical chemistry, urinalysis (rats only), histopathology, and reproductive system effects. The treatment concentrations were estimated to deliver daily doses of 0, 0.1, 0.2, 0.4, 0.6, 1.1 (males), or 0.8 (females) mg selenium/kg bw/day for rats and 0, 0.3, 0.5, 0.8, 1.5, or 2.6 mg/kg bw/day selenium for mice (ATSDR 1996). All male and female rats treated at the 60 mg/L level died within 11 and 6 weeks, respectively, while the mice were not affected at any of the concentrations. Growth of male and female mice and rats (prior to death) was reduced in the 30 and 60 mg/L treatments. The sodium selenate treatments were associated with increased incidences of renal papillary regeneration in rats starting at water concentrations of 7.5 mg/L. This may have been due to dehydration as water consumption also decreased with increasing selenium concentration. No lesions related to sodium selenate administration occurred in mice.

The National Toxicology Program (NTP 1994) conducted a similar study using sodium selenite. The same 13 week drinking water protocol was used, although, for this experiment the treatment levels were reduced to 0, 2, 4, 8, 16, or 32 mg/L. These concentrations were estimated to deliver daily doses of 0, 0.08, 0.13, 0.2, 0.4, 0.8 (males), or 0.9 (females) mg/kg bw/day selenium for rats and 0, 0.14, 0.3, 0.5, 0.9, or 1.6 mg/kg bw/day selenium for mice. The only mortality in this study was in the highest treatment group of female rats. Two died in this group and all other animals survived to the conclusion of the study. Weight loss was also experienced by rats and mice over the course of the study as body weights of those in the highest treatment group were reduced by 17 and 54%, respectively.

### ***Reproduction***

Acute and subacute exposures to selenium in feed and drinking water does not appear to affect the fertility of female animals unless the intake is sufficiently high to cause

general toxicity. In instances where the treatment levels are sufficiently high, general toxicity precludes any specific reproductive effects (Barlow and Sullivan 1982; Nobunaga *et al.* 1979). Chronic exposures have been shown to reduce fertility and to reduce the viability of the offspring of pairs that are able to conceive at doses somewhat below short term toxicity thresholds (Schroeder and Mitchener 1971b; Wahlstrom and Olson 1959; ATSDR 1996).

In a study conducted by Parshad (1999), albino Wistar rats were given daily intraperitoneal injections of sodium selenite at 2.0 or 4.0 mg/kg bw/day for 30 days. Animals in these groups experienced 14 and 40% mortality, respectively, over the course of the experiment and neither treatment had an effect on the length of the first two oestrus cycles. Examination of the ovaries at the conclusion of the experiment indicated that 21% of females in the low dose group and 60% of females in the high dose group had cystic follicles. Surviving animals with no cysts showed no signs of corpora lutea, indicating non-functional ovaries. In a subsequent study, the same investigators treated rats with intraperitoneal injections of 2 and 4 mg/kg bw/day for the 4 days of the oestrus cycle and then mated the females with fertile males. This procedure resulted in 12 and 28% mortality, respectively. Surviving females were sacrificed for examination on day 14 of gestation. The percent of females that conceived was reduced from 92 in controls to 73% in the low dose group and 50% in the high dose group. Significant reductions were also observed in the number of corpora lutea per female, the number of live embryos per litter and the number of implantation sites per litter.

Parshad and Sud (1989) have demonstrated that selenium is a reproductive toxicant to male rats. In their study, male Wistar rats were fed wheat grains that had naturally accumulated selenium to average levels of 12.5 mg/kg for 4 weeks. Rats in the treatment group were expected to have decreased food consumption and body weight

gain and so a third group of animals was underfed to compare results. As expected, the treatment and underfed groups were both undersized compared to controls and had significantly lower testis weights. There were no spermatozoa in the lumen of the seminiferous tubules in rats fed the wheat with naturally accumulated selenium.

Schroeder and Mitchener (1971b) conducted a three generation study in which CD mice were exposed to 3 mg/L sodium selenate in drinking water and 0.056 mg/kg in feed (estimated to be 0.76 mg selenium/kg bw/day; Sample *et al.* 1996) Five pairs of mice or rats were randomly selected for the first generation and subsequent generations were comprised of five pairs of the progeny of the previous generation. Control mice were bred for four generations with an average of 10-11 pups per litter. By the third generation of mice maintained on the selenium laced drinking water, only three litters were produced with an average of 7.6 pups per litter, compared with third generation controls which produced 22 litters with an average of 10.5 pups per litter. The total number of pups declined from 197 in the first generation to 169 in the second and 23 in the third. The number of runts per generation increased from 18% to 24% to 70% in the third generation, compared to less than 1% in all three generations of control animals.

Plasterer *et al.* (1985) treated female CD1 mice with sodium selenite at 7.0 mg/kg bw/day for 8 days. The dose level was selected as being just below the threshold of adult lethality and was administered on days 7-14 of gestation. Mice in this experiment showed no significant signs of reproductive toxicity in either total number of pregnant females, total number that delivered, or reproductive index.

### ***Field Surveys***

Selenium poisoning is a hazard to livestock in areas naturally rich in selenium (ATSDR 1996; Wilbur 1980; Rosenfeld and Beath 1964). “Blind staggers” is a

condition symptomatic of cattle and sheep grazing in such areas. Animals wander from the herd as their vision fails and, as the poisoning continues, their behavior becomes more erratic, limbs become weak, and the animals finally succumb to respiratory failure (Wilbur 1980). This effect in livestock is typically associated with Se concentrations of 400 to 800 mg/kg in plant material (Eisler 1985). Chronic exposures to selenium can lead to “alkali disease” which is characterized by growth retardation, inhibition of reproduction, hair loss, abnormal hoof formation, erosion of the cartilages, and degeneration of heart, kidney, and liver (Wilbur 1980). It has been postulated that Se displaces sulfur in keratin, resulting in structural changes in hair, nails, and hooves (Eisler 1985). Alkali disease is associated with the consumption of grains containing 5 - 40 mg selenium/kg over weeks or months (WHO 1987).

Due to concerns of atmospheric metal deposition and acid rain increasing metal mobilization, aquatic-dependent mammals were sampled from remote lakes in the Canadian Precambrian Shield (Wren 1984) for several metals. Selenium concentrations in otter and raccoon muscle tissue averaged approximately 0.2 mg/kg ww while concentrations in liver and kidney averaged 2-3 mg/kg ww. These tissue concentrations are approaching the criteria proposed by Eisler (2000) of 3-6 mg/kg for kidney and 12-15 mg/kg for liver as thresholds for protection against selenium toxicity.

The Kesterson National Wildlife Refuge in California suffered unusually high rates of embryonic mortality and abnormalities in the young of nesting aquatic birds in the early 1980s (Clark 1987). Tissue and media analysis revealed that high selenium concentrations may have contributed to the effects and irrigation drain water was identified as a source of the contamination. Clark (1987) sampled a number of small mammals in the Kesterson area and nearby reference areas and found no adverse

effects of selenium on wild mammals. Selenium concentrations in various mammal liver tissues ranged from a maximum of 250 mg/kg dw in the California vole to 0.91 mg/kg dw in the long-tailed weasel. The highest mammalian liver tissue concentration in the reference areas was 3.7 mg/kg dw in the house mouse. Clark *et al.* (1989) examined raccoons in the Kesterson Refuge and found liver tissue selenium concentrations of 19.9 mg/kg dw, but no evidence of selenium toxicity in any of a number of measurement endpoints. Rhian and Moxon (1943) noted inhibited growth in dogs associated with liver concentrations of 16-67 mg/kg dw and Rosenfeld and Beath (1964) reported that 4-32 mg/kg dw in livestock liver tissue was associated with “blind staggers” disease. Despite raccoons and voles surpassing these concentrations in the Kesterson Refuge, no pathologies were found in the animals.

### ***Effects Metrics***

Selenium toxicity is expressed over a narrow dose range with lethal and sublethal effects sharing the same dose range (ATSDR 1996). Studies in which reproductive effects were investigated found that for exposures in the range of one month, the fertility of female animals was unaffected, unless the intake was high enough to cause general toxicity (Barlow and Sullivan 1982; Nobunaga *et al.* 1979). Mortality is a more sensitive endpoint. The effects metrics for selenium are, therefore, based on the investigation conducted by Jacobs and Forst (1981) in which 5-week old female Sprague-Dawley rats were given water *ad libitum* treated with 1, 4, 8, 16, and 64 mg/L sodium selenite for 35 days. To convert these drinking water concentrations to daily doses, we multiplied the drinking water concentration by the daily water intake rate of 0.152 L/kg bw/day (TERA 2002). The estimated daily doses were 0.15, 0.61, 1.22, 2.43, and 9.73 mg/kg bw/day. Animals in the three lowest treatment groups suffered no significant mortalities, while the groups receiving the two highest doses suffered 80 and 100% mortality, respectively.

The dose-response relationship for female rat mortality versus selenium concentration in the diet was then estimated using the generalized linear model (GLiM) framework with a probit-link function for survival data (i.e., linear regression analysis on dose-response data that have had a probit transformation to linearize the relationship) and a binomial error distribution (Figure I2-7). Kerr and Meador (1996) and Bailer and Oris (1997) describe this framework in detail. The model parameters were  $\beta_0 = 3.08$ ,  $\beta_1 = -1.62$ ,  $se\beta_0 = 1.05$ ,  $se\beta_1 = 0.582$ , and  $corr\beta_0\beta_1 = -0.910$ . The resulting dose-response curve for the mortality effects of mercury to rats produced an adequate model to fit the data ( $F = 48067$ ;  $p < 0.0001$ ).

#### **2.2.2.4 Total PCBs**

Polychlorinated biphenyls (PCBs) is the generic term applied to a group of 209 chlorinated organic compounds that have similar molecular structures and properties. PCBs are persistent and highly lipophilic substances. Piscivorous mammals that reside, or partially reside, in the estuary are exposed to PCBs principally through diet and trophic transfer. PCBs are highly bioaccumulative substances that increase in concentration as they are passed up the food chain.

Studies have shown that PCBs are not particularly toxic during short term periods but that chronic exposure may result in effects at low levels (Coulston and Kolbye 1994; Aulerich *et al.* 1985; Bleavins *et al.* 1980).

#### ***Survival***

The greatest amount of toxicity information on total PCBs was found for Aroclor 1254. The prey tissue samples collected from the Calcasieu Estuary contained 50-60% chlorine. Thus, we focus the following discussion on studies that used higher

chlorinated mixtures (e.g., Aroclor 1254 and 1260). In a study where diet was prepared from cows that consumed feed contaminated with Aroclor 1254 (Platonow and Karstad 1973), a dose of 0.089 mg/kg bw/day over 160 days of exposure caused 100% mortality in the offspring of mink. The treatment also caused 17% mortality in adult females, but not in males.

Hornshaw *et al.* (1983) fed female mink with contaminated carp resulting in an exposure of 0.21 mg/kg bw/day Aroclor 1254. After 7 months of this feeding regime, the mink were allowed to breed and give birth to young. None of the young were born alive and 100% mortality in offspring was recorded. A slightly higher dose of 0.28 mg/kg bw/day in food of female mink caused 100% kit mortality 4 weeks after birth. Adult female mink experienced 12% mortality after 10 months of continuous exposure to this treatment (Aulerich and Ringer 1977). Total mortality in adults was observed at the dose of 0.50 mg/kg bw/day (Platonow and Karstad 1973). Only 105 days were required to kill the adults. In another study, female mink were exposed to a dose of 0.7 mg/kg bw/day, but only 30% of the individuals died after 9 months of exposure (Aulerich and Ringer 1977). The mortality increased to 71% at a dose of 1.4 mg/kg bw/day. Ranch mink exposed to 0.14 mg/kg bw/day Aroclor 1254 (from field-collected carp) experienced lower survival in lactating offspring (Wren *et al.* 1987b). The carp were collected from the field and contained other contaminants that could have contributed to the overall toxicity response. This concern has prompted us to eliminate this study from the derivation of a dose-response curve for PCBs.

Hornshaw *et al.* (1986) also conducted a 28 day study of acute oral toxicity to mink. The investigators used Aroclor 1254 as the toxicant, provided a constant exposure via the diet, and expressed toxicity in terms of concentration in diet. A daily dose can then be ascertained from the cumulative Aroclor consumption of the animals and their average body weight over the course of the study. Investigators conducting this study

noted a decrease in both feed consumption and body weight over 28 days. Mink fed a diet containing nominal concentrations of Aroclor 1254 of 58.3 mg/kg (estimated dose levels of 5.06 mg/kg bw/day for males and 8.43 mg/kg bw/day for females) experienced 50% mortality compared to all lower treatment levels, which showed no mortality.

Dietary LD<sub>50</sub> tests with mink performed by Hornshaw *et al.* (1986) revealed average LD<sub>50</sub>s that ranged from 6.58 mg/kg bw/day to 8.12 mg/kg bw/day. One of the highest estimates of mortality is reported by Aulerich *et al.* (1973), who estimated a 48-hr LD<sub>50</sub> of 140 mg/kg bw/day.

Dietary exposure of female mink to 0.004 mg/kg bw/day total PCBs (42 - 60 % chlorine) in carp for 3 to 6 weeks caused 15% mortality in kits (Heaton *et al.* 1995). The mortality increased to 69% at a level of 0.1 mg/kg bw/day after 3 weeks of exposure and 71% after 6 weeks of exposure.

Aulerich *et al.* (1985) fed adult female minks a diet of 2.5 mg/kg of Aroclor 1254. Ten percent of the mink died after 3 months of exposure. The female mink were mated with untreated males. Only 10% of the mated females whelped. In addition, a female gave birth to a single stillborn.

Kit mortality increased to 71% (3 weeks) and 89% (6 weeks) at a dose of 0.21 mg/kg bw/day (unspecified chlorine content; Heaton *et al.* 1995). Total kit mortality was observed at a dose of 0.36 mg/kg bw/day and deaths were observed in as little as 24 hours.

Jensen *et al.* (1977) exposed female mink to a dose of 1.54 mg/kg/day Aroclor 1254 for 66 days. As a result of the treatment, no live kits were born to any of the exposed

females. Ringer *et al.* (1972) exposed mink to a diet spiked with a total of 4.2 mg/kg bw/day PCB (equal amounts of Aroclors 1242, 1248, and 1254). All mink died prior to whelping.

The limited critical tissue residue data indicates that adverse effects begin to occur at around one milligram total PCBs in one kilogram of wet tissue weight. Heaton *et al.* (1995) reported a NOAEL of 0.09 mg/kg liver tissue. The LOAEL was 2.2 mg/kg liver. In another study, Leonards *et al.* (1995) reported an EC<sub>50</sub> of 1.2 mg/kg mink tissue for relative litter size. The EC<sub>50</sub> for kit survival was reported to be 2.4 mg/kg mink tissue. The study by Leonards *et al.* (1995) was a compilation of mink toxicity data for PCB technical mixtures containing 16 to 60% chlorine.

### ***Reproduction***

Ranch mink exposed to 0.14 mg/kg bw/day Aroclor 1254 experienced reduced survival in lactating offspring. However, no drop in fertility, whelping, or fecundity was observed (Wren *et al.* 1987b).

Kihlstrom *et al.* (1992) exposed female mink to 1.28 mg/kg bw/day in food for 105 days. The exposure caused all the kits to be stillborn. The dose also increased the number of interrupted pregnancies. Aulerich and Ringer (1977) reported that exposure of mink to 0.28 mg/kg bw/day does not lead to any observable effects on birth rate, birth weight, and survival. However, a dose of 2.8 mg/kg bw/day was associated with reduced whelping and growth rate of kits.

A dose of 0.004 mg/kg bw/day total PCBs in a diet of contaminated carp (42-50% chlorine) was associated with reduced growth rate in kits (Tillitt *et al.* 1996). Decreased fecundity was associated with an exposure to 0.08 mg/kg bw/day (0.700 mg/kg diet; Brunstrom *et al.* 1991).

Male and female mink fed PCB-contaminated diets (Saginaw Bay carp) had decreased breeding performance. Kit body weight and survival was reduced at birth at a dose of 0.14 mg/kg bw/day (Restum *et al.* 1998).

Jensen *et al.* (1977) exposed female mink to a dose of 1.54 mg/kg/day Aroclor 1254 for 66 days. As a result of the treatment, no live kits were born to any of the exposed females.

### ***Field Surveys***

No field surveys or studies were found in the literature involving the exposure of piscivorous mammals to total PCBs.

### ***Effects Metrics***

For the purpose of derivation of dose-response curves for total PCBs, we considered all studies that reported toxicity for Aroclor 1254 (see justification in Appendix G). The derivation of dose-response curves requires long-term feeding studies that report at least five dose levels on sensitive endpoints such as mortality or reproductive success. There were three studies that met these criteria. The studies included Hornshaw *et al.* (1983; six treatments), Hornshaw *et al.* (1986; 11 treatments), and Aulerich and Ringer (1977; six treatments). Four other studies reported dose-response data for mortality and fecundity, but had fewer than five dose levels. The studies were those performed by Platonow and Karstad (1973; two treatments), Aulerich *et al.* (1985; two treatments), and Heaton *et al.* (1995; four treatments). The derivation of a dose-response relationship for fecundity was further refined to include only those studies that used well-characterized diet (absence of field-collected fish), and used similar, state-of-the-art exposure protocols and laboratory facilities such as that used by the researchers at Michigan State University.

The acceptable studies that met the above criteria included those by Aulerich *et al.* (1985) and Aulerich and Ringer (1977). Because these studies used similar protocols, exposure duration, and species (similar strains), they were combined to yield a data set with 7 treatment levels for fecundity.

The dose-response relationship for fecundity versus PCBs concentration in the diet was then estimated using the generalized linear model (GLiM) framework with a log link function for number of fetus per female data (i.e., linear regression analysis on dose-response data that have had a log transformation to linearize the relationship) and a Poisson error distribution (Figure I2-8). Kerr and Meador (1996) and Bailer and Oris (1997) describe this framework in detail. The analysis was carried out in SAS® (SAS Institute, Cary, NC). The model parameters were  $\beta_0 = 1.62$ ,  $\beta_1 = -8.05$ ,  $se\beta_0 = 0.218$ ,  $se\beta_1 = 0.285$ , and  $corr\beta_0\beta_1 = -0.333$ . The resulting dose-response curve for fecundity of produced an adequate model fit to the data ( $P < 0.0001$ ; F value= 34.5).

### **2.2.3 Risk Characterization**

In the risk characterization phase of the probabilistic risk assessment, the results of the exposure assessment (i.e., reverse cumulative distribution functions) and effects assessment (i.e., dose-response relationships) were integrated to develop risk curves for each COC and each AOC. Ideally, risk characterization involves three major lines of evidence: comparison of modeled exposure to lab-derived effects metrics, *in situ* or whole-media toxicity tests; and, biological surveys. For piscivorous mammals, however, the latter two lines of evidence are not available. We therefore rely on the risk curves generated from the comparison of modeled exposure to laboratory derived dose-response curves.

## 3.0 Results

### 3.1 Probabilistic Exposure Assessment

#### *Mercury in Bayou d'Inde Area of Concern (BI AOC)*

The Monte Carlo analysis revealed that exposure of average-sized hypothetical piscivorous mammals to total mercury in the BI AOC of the Calcasieu Estuary could range from a minimum of 0.00978 to a maximum of 0.140 mg/kg bw/day. The mean exposure is 0.0315 mg/kg bw/day and the median exposure is 0.0296 mg/kg bw/day. Ninety percent of exposure estimates are between 0.0177 and 0.0524 mg/kg bw/day. For small piscivorous mammals, daily dose could range from a minimum of 0.0142 to a maximum of 0.180 mg/kg bw/day. The mean exposure is 0.0405 mg/kg bw/day and the median exposure is 0.0381 mg/kg bw/day. Ninety percent of exposure estimates are between 0.0241 and 0.0648 mg/kg bw/day. Figure I2-9 and Figure I2-10 depict the cumulative distributions of total mercury intake rates for the hypothetical average-sized and small piscivorous mammals.

Sensitivity analysis for the average-sized piscivorous mammals indicated that the power term used in the free metabolic rate equation was the most important variable [Pearson correlation coefficient ( $r_p$ ) = 0.64] followed by gross energy of fish ( $r_p$  = -0.59) and the slope term of the free metabolic rate equation ( $r_p$  = 0.40).

Sensitivity analysis for the small piscivorous mammals indicated that the gross energy of fish was the most important variable [Pearson correlation coefficient ( $r_p$ ) = -0.66] followed by the power term used in the free metabolic rate equation ( $r_p$  = 0.54) and the slope term of the free metabolic rate equation ( $r_p$  = 0.43).

The probability bounds analysis estimated for hypothetical average-sized piscivorous mammals foraging in the BI AOC of the Calcasieu Estuary are shown in Figure I2-9. The 10<sup>th</sup> percentile of the probability envelope formed by the lower and upper bounds ranges between 0.0129 and 0.0387 mg/kg bw/day. The 50<sup>th</sup> percentile ranges between 0.0198 and 0.0594 mg/kg bw/day, and the 90<sup>th</sup> percentile ranges between 0.0300 and 0.101 mg/kg bw/day. In comparison, the 10<sup>th</sup> percentile of the Monte Carlo distribution is 0.0196, the 50<sup>th</sup> percentile is 0.0294, and the 90<sup>th</sup> percentile is 0.0459 mg/kg bw/day. The probability bounds estimated for small piscivorous mammals are shown in Figure I2-10. The 10<sup>th</sup> percentile of the probability envelope formed by the lower and upper bounds ranges between 0.0170 and 0.0505 mg/kg bw/day. The 50<sup>th</sup> percentile ranges between 0.0255 and 0.0761 mg/kg bw/day, and the 90<sup>th</sup> percentile ranges between 0.0372 and 0.123 mg/kg bw/day. In comparison, the 10<sup>th</sup> percentile of the Monte Carlo distribution is 0.0265, the 50<sup>th</sup> percentile is 0.0381, and the 90<sup>th</sup> percentile is 0.0574 mg/kg bw/day.

### ***Mercury in Upper Calcasieu River Area of Concern (UCR AOC)***

The Monte Carlo analysis revealed that exposure of average-sized hypothetical piscivorous mammals to total mercury in the UCR AOC of the Calcasieu Estuary could range from a minimum of 0.00297 to a maximum of 0.0410 mg/kg bw/day. The mean exposure is 0.00956 mg/kg bw/day and the median exposure is 0.00897 mg/kg bw/day. Ninety percent of exposure estimates are between 0.00540 and 0.00896 mg/kg bw/day. For small piscivorous mammals, daily dose could range from a minimum of 0.00448 to a maximum of 0.0528 mg/kg bw/day. The mean exposure is 0.0123 mg/kg bw/day and the median exposure is 0.0116 mg/kg bw/day. Ninety percent of exposure estimates are between 0.00735 and 0.0196 mg/kg bw/day. Figure I2-11 and Figure I2-12 depict the cumulative distributions of total mercury intake rates for hypothetical average-sized and small piscivorous mammals.

Sensitivity analysis for the average-sized piscivorous mammals indicated that the power term used in the free metabolic rate equation was the most important variable [Pearson correlation coefficient ( $r_p$ ) = 0.64] followed by gross energy of fish ( $r_p$  = -0.59) and the slope term of the free metabolic rate equation ( $r_p$  = 0.40).

Sensitivity analysis for the small piscivorous mammals indicated that the gross energy of fish was the most important variable [Pearson correlation coefficient ( $r_p$ ) = -0.66] followed by the power term used in the free metabolic rate equation ( $r_p$  = 0.55) and the slope term of the free metabolic rate equation ( $r_p$  = 0.44).

The probability bounds analysis estimated for hypothetical average-sized piscivorous mammals foraging in the UCR AOC of the Calcasieu Estuary are shown in Figure I2-11. The 10<sup>th</sup> percentile of the probability envelope formed by the lower and upper bounds ranges between 0.00358 and 0.0123 mg/kg bw/day. The 50<sup>th</sup> percentile ranges between 0.00552 and 0.0196 mg/kg bw/day, and the 90<sup>th</sup> percentile ranges between 0.00823 and 0.0335 mg/kg bw/day. In comparison, the 10<sup>th</sup> percentile of the Monte Carlo distribution is 0.00598, the 50<sup>th</sup> percentile is 0.00896, and the 90<sup>th</sup> percentile is 0.0139 mg/kg bw/day. The probability bounds estimated for small piscivorous mammals are shown in Figure I2-12. The 10<sup>th</sup> percentile of the probability envelope formed by the lower and upper bounds ranges between 0.00479 and 0.0162 mg/kg bw/day. The 50<sup>th</sup> percentile ranges between 0.00716 and 0.0254 mg/kg bw/day, and the 90<sup>th</sup> percentile ranges between 0.0104 and 0.0421 mg/kg bw/day. In comparison, the 10<sup>th</sup> percentile of the Monte Carlo distribution is 0.00807, the 50<sup>th</sup> percentile is 0.00116, and the 90<sup>th</sup> percentile is 0.0174 mg/kg bw/day.

### ***Mercury in Middle Calcasieu River Area of Concern (MCR AOC)***

The Monte Carlo analysis revealed that exposure of average-sized hypothetical piscivorous mammals to total mercury in the MCR AOC of the Calcasieu Estuary

could range from a minimum of 0.00346 to a maximum of 0.0483 mg/kg bw/day. The mean exposure is 0.0112 mg/kg bw/day and the median exposure is 0.0106 mg/kg bw/day. Ninety percent of exposure estimates are between 0.00636 and 0.0186 mg/kg bw/day. For small piscivorous mammals, daily dose could range from a minimum of 0.00524 to a maximum of 0.0621 mg/kg bw/day. The mean exposure is 0.0144 mg/kg bw/day and the median exposure is 0.0136 mg/kg bw/day. Ninety percent of exposure estimates are between 0.00869 and 0.0230 mg/kg bw/day. Figure I2-13 and Figure I2-14 depict the cumulative distributions of total mercury intake rates for hypothetical average-sized and small piscivorous mammals.

Sensitivity analysis for the average-sized piscivorous mammals indicated that the power term used in the free metabolic rate equation was the most important variable [Pearson correlation coefficient ( $r_p$ ) = 0.65] followed by gross energy of fish ( $r_p$  = -0.59) and the slope term of the free metabolic rate equation ( $r_p$  = 0.40).

Sensitivity analysis for the small piscivorous mammals indicated that the gross energy of fish was the most important variable [Pearson correlation coefficient ( $r_p$ ) = -0.65] followed by the power term used in the free metabolic rate equation ( $r_p$  = 0.55) and the slope term of the free metabolic rate equation ( $r_p$  = 0.44).

The probability bounds analysis estimated for hypothetical average-sized piscivorous mammals foraging in the MCR AOC of the Calcasieu Estuary are shown in Figure I2-13. The 10<sup>th</sup> percentile of the probability envelope formed by the lower and upper bounds ranges between 0.00436 and 0.0152 mg/kg bw/day. The 50<sup>th</sup> percentile ranges between 0.00678 and 0.0246 mg/kg bw/day, and the 90<sup>th</sup> percentile ranges between 0.0101 and 0.0423 mg/kg bw/day. In comparison, the 10<sup>th</sup> percentile of the Monte Carlo distribution is 0.00703, the 50<sup>th</sup> percentile is 0.0106, and the 90<sup>th</sup> percentile is 0.0163 mg/kg bw/day. The probability bounds estimated for small piscivorous

mammals are shown in Figure I2-14. The 10<sup>th</sup> percentile of the probability envelope formed by the lower and upper bounds ranges between 0.00584 and 0.0201 mg/kg bw/day. The 50<sup>th</sup> percentile ranges between 0.00879 and 0.0317 mg/kg bw/day, and the 90<sup>th</sup> percentile ranges between 0.0128 and 0.0532 mg/kg bw/day. In comparison, the 10<sup>th</sup> percentile of the Monte Carlo distribution is 0.00950, the 50<sup>th</sup> percentile is 0.0136, and the 90<sup>th</sup> percentile is 0.0205 mg/kg bw/day.

### ***Mercury in Reference Areas***

The Monte Carlo analysis revealed that exposure of average-sized hypothetical piscivorous mammals to total mercury in the reference areas of the Calcasieu Estuary could range from a minimum of 0.00168 to a maximum of 0.0237 mg/kg bw/day. The mean exposure is 0.00546 mg/kg bw/day and the median exposure is 0.00512 mg/kg bw/day. Ninety percent of exposure estimates are between 0.00307 and 0.00905 mg/kg bw/day. For small piscivorous mammals, daily dose could range from a minimum of 0.00257 to a maximum of 0.0305 mg/kg bw/day. The mean exposure is 0.00701 mg/kg bw/day and the median exposure is 0.00661 mg/kg bw/day. Ninety percent of exposure estimates are between 0.00419 and 0.0112 mg/kg bw/day. Figure I2-15 and Figure I2-16 depict the cumulative distribution of total mercury intake rates for hypothetical average-sized and small piscivorous mammals.

Sensitivity analysis for the average-sized piscivorous mammals indicated that the power term used in the free metabolic rate equation was the most important variable [Pearson correlation coefficient ( $r_p$ ) = 0.64] followed by gross energy of fish ( $r_p$  = -0.60) and the slope term of the free metabolic rate equation ( $r_p$  = 0.39).

Sensitivity analysis for the small piscivorous mammals indicated that the gross energy of fish was the most important variable [Pearson correlation coefficient ( $r_p$ ) = -0.66]

followed by power term used in the free metabolic rate equation ( $r_p = 0.54$ ) and the slope term of the free metabolic rate equation ( $r_p = 0.44$ ).

The probability bounds analysis estimated for hypothetical average-sized piscivorous mammals foraging in the reference areas of the Calcasieu Estuary are shown in Figure I2-15. The 10<sup>th</sup> percentile of the probability envelope formed by the lower and upper bounds ranges between 0.00198 and 0.00764 mg/kg bw/day. The 50<sup>th</sup> percentile ranges between 0.00311 and 0.0126 mg/kg bw/day, and the 90<sup>th</sup> percentile ranges between 0.00468 and 0.0221 mg/kg bw/day. In comparison, the 10<sup>th</sup> percentile of the Monte Carlo distribution is 0.00340, the 50<sup>th</sup> percentile is 0.00513, and the 90<sup>th</sup> percentile is 0.00798 mg/kg bw/day. The probability bounds estimated for small piscivorous mammals are shown in Figure I2-16. The 10<sup>th</sup> percentile of the probability envelope formed by the lower and upper bounds ranges between 0.00266 and 0.0101 mg/kg bw/day. The 50<sup>th</sup> percentile ranges between 0.00404 and 0.0163 mg/kg bw/day, and the 90<sup>th</sup> percentile ranges between 0.00592 and 0.0278 mg/kg bw/day. In comparison, the 10<sup>th</sup> percentile of the Monte Carlo distribution is 0.00460, the 50<sup>th</sup> percentile is 0.00660, and the 90<sup>th</sup> percentile is 0.00998 mg/kg bw/day.

#### ***TCDD-TEQs in Bayou d'Inde Area of Concern (BI AOC)***

The Monte Carlo analysis revealed that exposure of average-sized hypothetical piscivorous mammals to TCDD-TEQs in the BI AOC of the Calcasieu Estuary could range from a minimum of 2.42 to a maximum of 41.2 ng/kg bw/day. The mean exposure is 7.86 ng/kg bw/day and the median exposure is 7.38 ng/kg bw/day. Ninety percent of exposure estimates are between 4.44 and 12.8 ng/kg bw/day. For small piscivorous mammals, daily dose could range from a minimum of 3.60 to a maximum of 49.4 ng/kg bw/day. The mean exposure is 10.1 ng/kg bw/day and the median exposure is 9.56 ng/kg bw/day. Ninety percent of exposure estimates are between 6.05 and 15.9 ng/kg bw/day. Figure I2-17 and Figure I2-18 depict the cumulative

distributions of TCDD-TEQs intake rates for hypothetical average-sized and small piscivorous mammals.

Sensitivity analysis for the average-sized piscivorous mammals indicated that the power term used in the free metabolic rate equation was the most important variable [Pearson correlation coefficient ( $r_p$ ) = 0.64] followed by gross energy of fish ( $r_p$  = -0.59) and the slope term of the free metabolic rate equation ( $r_p$  = 0.39).

Sensitivity analysis for the small piscivorous mammals indicated that the gross energy of fish was the most important variable [Pearson correlation coefficient ( $r_p$ ) = -0.64] followed by the power term used in the free metabolic rate equation ( $r_p$  = 0.55) and the slope term of the free metabolic rate equation ( $r_p$  = 0.43).

The probability bounds analysis estimated for hypothetical average-sized piscivorous mammals foraging in the BI AOC of the Calcasieu Estuary are shown in Figure I2-17. The 10<sup>th</sup> percentile of the probability envelope formed by the lower and upper bounds ranges between 2.14 and 16.7 ng/kg bw/day. The 50<sup>th</sup> percentile ranges between 3.60 and 26.7 ng/kg bw/day, and the 90<sup>th</sup> percentile ranges between 5.86 and 47.7 ng/kg bw/day. In comparison, the 10<sup>th</sup> percentile of the Monte Carlo distribution is 4.96, the 50<sup>th</sup> percentile is 7.39, and the 90<sup>th</sup> percentile is 11.2 ng/kg bw/day. The probability bounds estimated for small piscivorous mammals are shown in Figure I2-18. The 10<sup>th</sup> percentile of the probability envelope formed by the lower and upper bounds ranges between 2.85 and 22.1 ng/kg bw/day. The 50<sup>th</sup> percentile ranges between 4.66 and 34.4 ng/kg bw/day, and the 90<sup>th</sup> percentile ranges between 7.44 and 60.2 ng/kg bw/day. In comparison, the 10<sup>th</sup> percentile of the Monte Carlo distribution is 6.67, the 50<sup>th</sup> percentile is 9.54, and the 90<sup>th</sup> percentile is 14.0 ng/kg bw/day.

***TCDD-TEQs in Upper Calcasieu River Area of Concern (UCR AOC)***

The Monte Carlo analysis revealed that exposure of average-sized hypothetical piscivorous mammals to TCDD-TEQs in the UCR AOC of the Calcasieu Estuary could range from a minimum of 0.798 to a maximum of 14.3 ng/kg bw/day. The mean exposure is 2.67 ng/kg bw/day and the median exposure is 2.52 ng/kg bw/day. Ninety percent of exposure estimates are between 1.50 and 4.38 ng/kg bw/day. For small piscivorous mammals, daily dose could range from a minimum of 1.19 to a maximum of 17.1 ng/kg bw/day. The mean exposure is 3.43 ng/kg bw/day and the median exposure is 3.24 ng/kg bw/day. Ninety percent of exposure estimates are between 2.06 and 5.50 ng/kg bw/day. Figure I2-19 and Figure I2-20 depict the cumulative distributions of TCDD-TEQs intake rates for hypothetical average-sized and small piscivorous mammals.

Sensitivity analysis for the average-sized piscivorous mammals indicated that the power term used in the free metabolic rate equation was the most important variable [Pearson correlation coefficient ( $r_p$ ) = 0.64] followed by gross energy of fish ( $r_p$  = -0.58) and the slope term of the free metabolic rate equation ( $r_p$  = 0.39).

Sensitivity analysis for the small piscivorous mammals indicated that the gross energy of fish was the most important variable [Pearson correlation coefficient ( $r_p$ ) = -0.65] followed by the power term used in the free metabolic rate equation ( $r_p$  = 0.55) and the slope term of the free metabolic rate equation ( $r_p$  = 0.43).

The probability bounds analysis estimated for hypothetical average-sized piscivorous mammals foraging in the UCR AOC of the Calcasieu Estuary are shown in Figure I2-19. The 10<sup>th</sup> percentile of the probability envelope formed by the lower and upper bounds ranges between 0.505 and 6.21 ng/kg bw/day. The 50<sup>th</sup> percentile ranges between 0.958 and 9.48 ng/kg bw/day, and the 90<sup>th</sup> percentile ranges between 1.77

and 16.6 ng/kg bw/day. In comparison, the 10<sup>th</sup> percentile of the Monte Carlo distribution is 1.69, the 50<sup>th</sup> percentile is 2.51, and the 90<sup>th</sup> percentile is 3.86 ng/kg bw/day. The probability bounds estimated for small piscivorous mammals are depicted in Figure I2-20. The 10<sup>th</sup> percentile of the probability envelope formed by the lower and upper bounds ranges between 0.668 and 8.25 ng/kg bw/day. The 50<sup>th</sup> percentile ranges between 1.24 and 12.2 ng/kg bw/day, and the 90<sup>th</sup> percentile ranges between 2.26 and 21.0 ng/kg bw/day. In comparison, the 10<sup>th</sup> percentile of the Monte Carlo distribution is 2.27, the 50<sup>th</sup> percentile is 3.24, and the 90<sup>th</sup> percentile is 4.79 ng/kg bw/day.

#### ***TCDD-TEQs in Middle Calcasieu River Area of Concern (MCR AOC)***

The Monte Carlo analysis revealed that exposure of average-sized hypothetical piscivorous mammals to TCDD-TEQs in the MCR AOC of the Calcasieu Estuary could range from a minimum of 0.893 to a maximum of 10.9 ng/kg bw/day. The mean exposure is 3.23 ng/kg bw/day and the median exposure is 3.08 ng/kg bw/day. Ninety percent of exposure estimates are between 1.82 and 5.19 ng/kg bw/day. For small piscivorous mammals, daily dose could range from a minimum of 1.31 to a maximum of 12.74 ng/kg bw/day. The mean exposure is 4.15 ng/kg bw/day and the median exposure is 3.98 ng/kg bw/day. Ninety percent of exposure estimates are between 2.48 and 6.41 ng/kg bw/day. Figure I2-21 and Figure I2-22 depict the cumulative distributions of TCDD-TEQs intake rates for hypothetical average-sized and small piscivorous mammals.

Sensitivity analysis for the average-sized piscivorous mammals indicated that the power term used in the free metabolic rate equation was the most important variable [Pearson correlation coefficient ( $r_p$ ) = 0.66] followed by the gross energy of fish ( $r_p$  = -0.57) and the slope term of the free metabolic rate equation ( $r_p$  = 0.40).

Sensitivity analysis for the small piscivorous mammals indicated that the gross energy of fish was the most important variable [Pearson correlation coefficient ( $r_p$ ) = -0.63] followed by the power term used in the free metabolic rate equation ( $r_p$  = 0.56) and the slope term of the free metabolic rate equation ( $r_p$  = 0.44).

The probability bounds analysis estimated for hypothetical average-sized piscivorous mammals foraging in the MCR AOC of the Calcasieu Estuary are shown in Figure I2-21. The 10<sup>th</sup> percentile of the probability envelope formed by the lower and upper bounds ranges between 0.326 and 6.27 ng/kg bw/day. The 50<sup>th</sup> percentile ranges between 0.493 and 9.14 ng/kg bw/day, and the 90<sup>th</sup> percentile ranges between 0.724 and 14.4 ng/kg bw/day. In comparison, the 10<sup>th</sup> percentile of the Monte Carlo distribution is 2.04, the 50<sup>th</sup> percentile is 3.08, and the 90<sup>th</sup> percentile is 4.61 ng/kg bw/day. The probability bounds estimated for small piscivorous mammals are depicted in Figure I2-22. The 10<sup>th</sup> percentile of the probability envelope formed by the lower and upper bounds ranges between 0.438 and 8.34 ng/kg bw/day. The 50<sup>th</sup> percentile ranges between 0.639 and 11.8 ng/kg bw/day, and the 90<sup>th</sup> percentile ranges between 0.912 and 18.0 ng/kg bw/day. In comparison, the 10<sup>th</sup> percentile of the Monte Carlo distribution is 2.75, the 50<sup>th</sup> percentile is 3.98, and the 90<sup>th</sup> percentile is 5.74 ng/kg bw/day.

### ***TCDD-TEQs in Reference Areas***

The Monte Carlo analysis revealed that exposure of average-sized hypothetical piscivorous mammals to TCDD and equivalents in the reference areas of the Calcasieu Estuary could range from a minimum of 0.527 to a maximum of 9.98 ng/kg bw/day. The mean exposure is 1.81 ng/kg bw/day and the median exposure is 1.69 ng/kg bw/day. Ninety percent of exposure estimates are between 1.01 and 3.00 ng/kg bw/day. For small piscivorous mammals, daily dose could range from a minimum of 0.784 to a maximum of 11.9 ng/kg bw/day. The mean exposure is 2.32 ng/kg bw/day

and the median exposure is 2.19 ng/kg bw/day. Ninety percent of exposure estimates are between 1.37 and 3.72 ng/kg bw/day. Figure I2-23 and Figure I2-24 depict the cumulative distributions of TCDD-TEQs intake rates for hypothetical average-sized and small piscivorous mammals.

Sensitivity analysis for the average-sized piscivorous mammals indicated that the power term used in the free metabolic rate equation was the most important variable [Pearson correlation coefficient ( $r_p$ ) = 0.63] followed by gross energy of fish ( $r_p$  = -0.60) and the slope term of the free metabolic rate equation ( $r_p$  = 0.38).

Sensitivity analysis for the small piscivorous mammals indicated that the gross energy of fish was the most important variable [Pearson correlation coefficient ( $r_p$ ) = -0.67] followed by the power term used in the free metabolic rate equation ( $r_p$  = 0.54) and the slope term of the free metabolic rate equation ( $r_p$  = 0.42).

The probability bounds analysis estimated for hypothetical average-sized piscivorous mammals foraging in the reference areas of the Calcasieu Estuary are shown in Figure I2-23. The 10<sup>th</sup> percentile of the probability envelope formed by the lower and upper bounds ranges between 0.653 and 2.07 ng/kg bw/day. The 50<sup>th</sup> percentile ranges between 0.999 and 3.02 ng/kg bw/day, and the 90<sup>th</sup> percentile ranges between 1.48 and 4.79 ng/kg bw/day. In comparison, the 10<sup>th</sup> percentile of the Monte Carlo distribution is 1.13, the 50<sup>th</sup> percentile is 1.69, and the 90<sup>th</sup> percentile is 2.62 ng/kg bw/day. The probability bounds estimated for small piscivorous mammals are depicted in Figure I2-24. The 10<sup>th</sup> percentile of the probability envelope formed by the lower and upper bounds ranges between 0.876 and 2.75 ng/kg bw/day. The 50<sup>th</sup> percentile ranges between 1.29 and 3.90 ng/kg bw/day, and the 90<sup>th</sup> percentile ranges between

1.87 and 6.00 ng/kg bw/day. In comparison, the 10<sup>th</sup> percentile of the Monte Carlo distribution is 1.52, the 50<sup>th</sup> percentile is 2.20, and the 90<sup>th</sup> percentile is 3.26 ng/kg bw/day.

***Selenium in Bayou d’Inde Area of Concern (BI AOC)***

The Monte Carlo analysis revealed that exposure of average-sized hypothetical piscivorous mammals to selenium in the BI AOC of the Calcasieu Estuary could range from a minimum of 0.0288 to a maximum of 0.412 mg/kg bw/day. The mean exposure is 0.107 mg/kg bw/day and the median exposure is 0.100 mg/kg bw/day. Ninety percent of exposure estimates are between 0.0615 and 0.176 mg/kg bw/day. For small piscivorous mammals, daily dose could range from a minimum of 0.0430 to a maximum of 0.488 mg/kg bw/day. The mean exposure is 0.137 mg/kg bw/day and the median exposure is 0.130 mg/kg bw/day. Ninety percent of exposure estimates are between 0.0834 and 0.217 mg/kg bw/day. Figure I2-25 and Figure I2-26 depict the cumulative distributions of selenium intake rates for hypothetical average-sized and small piscivorous mammals.

Sensitivity analysis for the average-sized piscivorous mammals indicated that the power term used in the free metabolic rate equation was the most important variable [Pearson correlation coefficient ( $r_p$ ) = 0.66] followed by gross energy of fish ( $r_p$  = -0.56) and the slope term of the free metabolic rate equation ( $r_p$  = 0.40).

Sensitivity analysis for the small piscivorous mammals indicated that the gross energy of fish was the most important variable [Pearson correlation coefficient ( $r_p$ ) = -0.62] followed by the power term used in the free metabolic rate equation ( $r_p$  = 0.56) and the slope term of the free metabolic rate equation ( $r_p$  = 0.44).

The probability bounds analysis estimated for hypothetical averaged-sized piscivorous mammal foraging in the BI AOC of the Calcasieu Estuary are shown in Figure I2-25. The 10<sup>th</sup> percentile of the probability envelope formed by the lower and upper bounds ranges between 0.0474 and 0.141 mg/kg bw/day. The 50<sup>th</sup> percentile ranges between 0.0717 and 0.217 mg/kg bw/day, and the 90<sup>th</sup> percentile ranges between 0.108 and 0.366 mg/kg bw/day. In comparison, the 10<sup>th</sup> percentile of the Monte Carlo distribution is 0.0679, the 50<sup>th</sup> percentile is 0.101, and the 90<sup>th</sup> percentile is 0.154 mg/kg bw/day. The probability bounds estimated for small piscivorous mammals are shown in Figure I2-26. The 10<sup>th</sup> percentile of the probability envelope formed by the lower and upper bounds ranges between 0.0634 and 0.186 mg/kg bw/day. The 50<sup>th</sup> percentile ranges between 0.0929 and 0.280 mg/kg bw/day, and the 90<sup>th</sup> percentile ranges between 0.1360 and 0.459 mg/kg bw/day. In comparison, the 10<sup>th</sup> percentile of the Monte Carlo distribution is 0.0921, the 50<sup>th</sup> percentile is 0.130, and the 90<sup>th</sup> percentile is 0.191 mg/kg bw/day.

#### ***Selenium in Middle Calcasieu River Area of Concern (MCR AOC)***

The Monte Carlo analysis revealed that exposure of average-sized hypothetical piscivorous mammals to selenium in the MCR AOC of the Calcasieu Estuary could range from a minimum of 0.0307 to a maximum of 0.435 mg/kg bw/day. The mean exposure is 0.113 mg/kg bw/day and the median exposure is 0.106 mg/kg bw/day. Ninety percent of exposure estimates are between 0.0647 and 0.186 mg/kg bw/day. For small piscivorous mammals, daily dose could range from a minimum of 0.0458 to a maximum of 0.517 mg/kg bw/day. The mean exposure is 0.145 mg/kg bw/day and the median exposure is 0.137 mg/kg bw/day. Ninety percent of exposure estimates are between 0.0877 and 0.229 mg/kg bw/day. Figure I2-27 and Figure I2-28 depict the cumulative distributions of selenium intake rates for hypothetical average-sized and small piscivorous mammals.

Sensitivity analysis for the average-sized piscivorous mammals indicated that the power term used in the free metabolic rate equation was the most important variable [Pearson correlation coefficient ( $r_p$ ) = 0.66] followed by gross energy of fish ( $r_p$  = -0.55) and the slope term of the free metabolic rate equation ( $r_p$  = 0.39).

Sensitivity analysis for the small piscivorous mammals indicated that the gross energy of fish was the most important variable [Pearson correlation coefficient ( $r_p$ ) = -0.62] followed by the power term used in the free metabolic rate equation ( $r_p$  = 0.56) and the slope term of the free metabolic rate equation ( $r_p$  = 0.44).

The probability bounds analysis estimated for hypothetical average-sized piscivorous mammals foraging in the MCR AOC of the Calcasieu Estuary are shown in Figure I2-27. The 10<sup>th</sup> percentile of the probability envelope formed by the lower and upper bounds ranges between 0.0407 and 0.159 mg/kg bw/day. The 50<sup>th</sup> percentile ranges between 0.0627 and 0.260 mg/kg bw/day, and the 90<sup>th</sup> percentile ranges between 0.0955 and 0.467 mg/kg bw/day. In comparison, the 10<sup>th</sup> percentile of the Monte Carlo distribution is 0.0720, the 50<sup>th</sup> percentile is 0.106, and the 90<sup>th</sup> percentile is 0.162 mg/kg bw/day. The probability bounds estimated for small piscivorous mammals are shown in Figure I2-28. The 10<sup>th</sup> percentile of the probability envelope formed by the lower and upper bounds ranges between 0.0544 and 0.209 mg/kg bw/day. The 50<sup>th</sup> percentile ranges between 0.0813 and 0.336 mg/kg bw/day, and the 90<sup>th</sup> percentile ranges between 0.120 and 0.586 mg/kg bw/day. In comparison, the 10<sup>th</sup> percentile of the Monte Carlo distribution is 0.0971, the 50<sup>th</sup> percentile is 0.137, and the 90<sup>th</sup> percentile is 0.202 mg/kg bw/day.

### ***Selenium in Reference Areas***

The Monte Carlo analysis revealed that exposure of average-sized hypothetical piscivorous mammals to selenium in the reference areas of the Calcasieu Estuary could

range from a minimum of 0.0221 to a maximum of 0.308 mg/kg bw/day. The mean exposure is 0.0813 mg/kg bw/day and the median exposure is 0.0765 mg/kg bw/day. Ninety percent of exposure estimates are between 0.0469 and 0.134 mg/kg bw/day. For small piscivorous mammals, daily dose could range from a minimum of 0.0333 to a maximum of 0.366 mg/kg bw/day. The mean exposure is 0.104 mg/kg bw/day and the median exposure is 0.0991 mg/kg bw/day. Ninety percent of exposure estimates are between 0.0637 and 0.164 mg/kg bw/day. Figure I2-29 and Figure I2-30 depict the cumulative distributions of selenium intake rates for hypothetical average-sized and small piscivorous mammals.

Sensitivity analysis for the average-sized piscivorous mammals indicated that the power term used in the free metabolic rate equation was the most important variable [Pearson correlation coefficient ( $r_p$ ) = 0.66] followed by gross energy of fish ( $r_p$  = -0.54) and the slope term of the free metabolic rate equation ( $r_p$  = 0.40).

Sensitivity analysis for the small piscivorous mammals indicated that the gross energy of fish was the most important variable [Pearson correlation coefficient ( $r_p$ ) = -0.61] followed by the power term used in the free metabolic rate equation ( $r_p$  = 0.57) and the slope term of the free metabolic rate equation ( $r_p$  = 0.44).

The probability bounds analysis estimated for hypothetical average-sized piscivorous mammals foraging in the reference areas of the Calcasieu Estuary are shown in Figure I2-29. The 10<sup>th</sup> percentile of the probability envelope formed by the lower and upper bounds ranges between 0.0293 and 0.120 mg/kg bw/day. The 50<sup>th</sup> percentile ranges between 0.0452 and 0.200 mg/kg bw/day, and the 90<sup>th</sup> percentile ranges between 0.0689 and 0.365 mg/kg bw/day. In comparison, the 10<sup>th</sup> percentile of the Monte Carlo distribution is 0.0519, the 50<sup>th</sup> percentile is 0.0765, and the 90<sup>th</sup> percentile is 0.116 mg/kg bw/day. The probability bounds estimated for small piscivorous

mammals are shown in Figure I2-30. The 10<sup>th</sup> percentile of the probability envelope formed by the lower and upper bounds ranges between 0.0392 and 0.158 mg/kg bw/day. The 50<sup>th</sup> percentile ranges between 0.0586 and 0.259 mg/kg bw/day, and the 90<sup>th</sup> percentile ranges between 0.0869 and 0.459 mg/kg bw/day. In comparison, the 10<sup>th</sup> percentile of the Monte Carlo distribution is 0.0700, the 50<sup>th</sup> percentile is 0.0993, and the 90<sup>th</sup> percentile is 0.145 mg/kg bw/day.

***PCBs in Bayou d'Inde Area of Concern (BI AOC)***

The Monte Carlo analysis revealed that exposure of average-sized hypothetical piscivorous mammals to PCBs in the BI AOC of the Calcasieu Estuary could range from a minimum of 0.0110 to a maximum of 0.151 mg/kg bw/day. The mean exposure is 0.0400 mg/kg bw/day and the median exposure is 0.0375 mg/kg bw/day. Ninety percent of exposure estimates are between 0.0221 and 0.0665 mg/kg bw/day. For small piscivorous mammals, daily dose could range from a minimum of 0.0162 to a maximum of 0.182 mg/kg bw/day. The mean exposure is 0.0513 mg/kg bw/day and the median exposure is 0.0484 mg/kg bw/day. Ninety percent of exposure estimates are between 0.0301 and 0.0484 mg/kg bw/day. Figure I2-31 and Figure I2-32 depict the cumulative distributions of PCBs intake rates for hypothetical average-sized and small piscivorous mammals.

Sensitivity analysis for the average-sized piscivorous mammals indicated that the power term used in the free metabolic rate equation was the most important variable [Pearson correlation coefficient ( $r_p$ ) = 0.63] followed by gross energy of fish ( $r_p$  = -0.59) and the slope term of the free metabolic rate equation ( $r_p$  = 0.39).

Sensitivity analysis for the small piscivorous mammals indicated that the gross energy of fish was the most important variable [Pearson correlation coefficient ( $r_p$ ) = -0.65]

followed by the power term used in the free metabolic rate equation ( $r_p = 0.54$ ) and the slope term of the free metabolic rate equation ( $r_p = 0.43$ ).

The probability bounds analysis estimated for hypothetical average-sized piscivorous mammals foraging in the BI AOC of the Calcasieu Estuary are shown in Figure I2-31. The 10<sup>th</sup> percentile of the probability envelope formed by the lower and upper bounds ranges between 0.0119 and 0.0565 mg/kg bw/day. The 50<sup>th</sup> percentile ranges between 0.0187 and 0.0965 mg/kg bw/day, and the 90<sup>th</sup> percentile ranges between 0.0289 and 0.187 mg/kg bw/day. In comparison, the 10<sup>th</sup> percentile of the Monte Carlo distribution is 0.0248, the 50<sup>th</sup> percentile is 0.0375, and the 90<sup>th</sup> percentile is 0.0580 mg/kg bw/day. The probability bounds estimated for small piscivorous mammals are shown in Figure I2-32. The 10<sup>th</sup> percentile of the probability envelope formed by the lower and upper bounds ranges between 0.0158 and 0.0743 mg/kg bw/day. The 50<sup>th</sup> percentile ranges between 0.0243 and 0.125 mg/kg bw/day, and the 90<sup>th</sup> percentile ranges between 0.0365 and 0.235 mg/kg bw/day. In comparison, the 10<sup>th</sup> percentile of the Monte Carlo distribution is 0.0337, the 50<sup>th</sup> percentile is 0.0485, and the 90<sup>th</sup> percentile is 0.0726 mg/kg bw/day.

### ***PCBs in Reference Areas***

The Monte Carlo analysis revealed that exposure of average-sized hypothetical piscivorous mammals to PCBs in the reference areas of the Calcasieu Estuary could range from a minimum of 0.00171 to a maximum of 0.0216 mg/kg bw/day. The mean exposure is 0.00594 mg/kg bw/day and the median exposure is 0.00556 mg/kg bw/day. Ninety percent of exposure estimates are between 0.00330 and 0.00982 mg/kg bw/day. For small piscivorous mammals, daily dose could range from a minimum of 0.00251 to a maximum of 0.0257 mg/kg bw/day. The mean exposure is 0.00762 mg/kg bw/day and the median exposure is 0.00720 mg/kg bw/day. Ninety percent of exposure estimates are between 0.00452 and 0.0121 mg/kg bw/day. Figure

I2-33 and Figure I2-34 depict the cumulative distributions of PCBs intake rates for hypothetical average-sized and small piscivorous mammals.

Sensitivity analysis for the average-sized piscivorous mammals indicated that the power term used in the free metabolic rate equation was the most important variable [Pearson correlation coefficient ( $r_p$ ) = 0.64] followed by gross energy of fish ( $r_p$  = -0.58) and the slope term of the free metabolic rate equation ( $r_p$  = 0.40).

Sensitivity analysis for the small piscivorous mammals indicated that the gross energy of fish was the most important variable [Pearson correlation coefficient ( $r_p$ ) = -0.64] followed by the power term used in the free metabolic rate equation ( $r_p$  = 0.54) and the slope term of the free metabolic rate equation ( $r_p$  = 0.44).

The probability bounds analysis estimated for hypothetical average-sized piscivorous mammals foraging in the reference areas of the Calcasieu Estuary are shown in Figure I2-33. The 10<sup>th</sup> percentile of the probability envelope formed by the lower and upper bounds ranges between 0.00186 and 0.00933 mg/kg bw/day. The 50<sup>th</sup> percentile ranges between 0.00332 and 0.0158 mg/kg bw/day, and the 90<sup>th</sup> percentile ranges between 0.00562 and 0.0300 mg/kg bw/day. In comparison, the 10<sup>th</sup> percentile of the Monte Carlo distribution is 0.00373, the 50<sup>th</sup> percentile is 0.00556, and the 90<sup>th</sup> percentile is 0.00859 mg/kg bw/day. The probability bounds estimated for small piscivorous mammals are shown in Figure I2-34. The 10<sup>th</sup> percentile of the probability envelope formed by the lower and upper bounds ranges between 0.00246 and 0.0123 mg/kg bw/day. The 50<sup>th</sup> percentile ranges between 0.00432 and 0.0204 mg/kg bw/day, and the 90<sup>th</sup> percentile ranges between 0.00714 and 0.0380 mg/kg bw/day. In comparison, the 10<sup>th</sup> percentile of the Monte Carlo distribution is 0.00501, the 50<sup>th</sup> percentile is 0.00720, and the 90<sup>th</sup> percentile is 0.0107 mg/kg bw/day.

## 3.2 Risk Assessment

The effects characterization (Section 2.2.2) provided a detailed overview of the current toxicological literature on the potential effects of mercury, TCDD-TEQs, selenium and total PCBs to mammalian species. Studies were available to directly quantify the effects of mercury and total PCBs to piscivorous mammals. For TCDD-TEQs and selenium, surrogate mammal species were used. In all cases, there were sufficient data to develop dose-response curves to characterize effects.

The following discussion presents an integration of the exposure distributions and effects curves developed for average-sized and small piscivorous mammals for each exposure area. The categories of low, indeterminate, and high risk were derived using the following guidance:

- If the probability of 10% or greater effect was less than 20%, then the risk to mammals was low;
- If the probability of 20% or greater effect was greater than 50%, then the risk to mammals was high; and,
- All other outcomes were considered to have indeterminate risk.

### ***Mercury in Bayou d'Inde Area of Concern (BI AOC)***

The effects metric for mercury was derived by combining the results of Aulerich *et al.* 1974; Wobeser *et al.* 1976; Wren *et al.* 1987a; Chamberland *et al.* 1996; and Dansereau *et al.* 1999. The toxicological endpoint was mortality to female mink. The dose-response curve indicated that 10% and 20% declines in mortality would be expected at doses of 0.0952 and 0.114 mg/kg bw/day, respectively.

Integration of the methylmercury effects curve with the Monte Carlo curve for the exposure of the average-sized piscivorous mammals to mercury in the BI AOC of the Calcasieu Estuary indicates that there is a 0% probability of exceeding the 10% and 20% effects doses (Table I2-4). The probability bounds analysis indicates that the probability of exceeding the 10% effect dose could range from 0% to 12%, given the uncertainties that exist about how to parameterize the Monte Carlo exposure model. The corresponding probability of exceeding the 20% effects doses could range from 0% to 7%. Figure I2-35 shows the estimated probabilities of effects of differing magnitude for average-sized piscivorous mammals in BI AOC. Thus, average-sized piscivorous mammals feeding in the BI AOC are at a low risk of toxicity from exposure to mercury (Table I2-4).

The Monte Carlo analysis indicates that average-sized piscivorous mammals have a 100% probability of total daily mercury intake exceeding the Appendix G benchmark of 0.0116 mg/kg bw/day. The lower and upper probability bounds of exposure have a 95% and 100% probability of exceeding the Appendix G benchmark, respectively. The probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I2-5.

Integration of the methylmercury effects curve with the Monte Carlo curve for the exposure of the small piscivorous mammals to mercury in the BI AOC of the Calcasieu Estuary indicates that there is a 0% probability of exceeding the 10% and 20% effects doses (Table I2-4). The probability bounds analysis indicates that the probability of exceeding the 10% effect dose could range from 0% to 26%, given the uncertainties that exist about how to parameterize the Monte Carlo exposure model. The corresponding probability of exceeding the 20% effects doses could range from 0% to 14%. Figure I2-36 shows the estimated probabilities of effects of differing magnitude for small piscivorous mammals in BI AOC. Thus, small piscivorous

mammals feeding in the BI AOC are at a low risk of toxicity from exposure to mercury (Table I2-4).

The Monte Carlo analysis indicates that small piscivorous mammals have a 100% probability of total daily mercury intake exceeding the Appendix G benchmark of 0.0116 mg/kg bw/day. The lower and upper probability bounds of exposure have a 99% and 100% probability of exceeding the Appendix G benchmark, respectively. The probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I2-5.

***Mercury in Upper Calcasieu River Area of Concern (UCR AOC)***

Integration of the methylmercury effects curve with the Monte Carlo curve for the exposure of the average-sized piscivorous mammals to mercury in the UCR AOC of the Calcasieu Estuary indicates that there is a 0% probability of exceeding the 10% and 20% effects doses (Table I2-4). The probability bounds analysis indicates that the probability of exceeding the 10% and 20% effects doses is 0%, despite the uncertainties that exist about how to parameterize the Monte Carlo exposure model. Thus, average-sized piscivorous mammals feeding in the UCR AOC are at a low risk of toxicity from exposure to mercury (Table I2-4).

The Monte Carlo analysis indicates that average-sized piscivorous mammals have a 21.9% probability of total daily mercury intake exceeding the Appendix G benchmark of 0.0116 mg/kg bw/day. The lower and upper probability bounds of exposure have a 0% and 93% probability of exceeding the Appendix G benchmark, respectively. The probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I2-5.

Integration of the methylmercury effects curve with the Monte Carlo curve for the exposure of the small piscivorous mammals to mercury in the UCR AOC of the Calcasieu Estuary indicates that there is a 0% probability of exceeding the 10% and 20% effects doses (Table I2-4). The probability bounds analysis indicates that the probability of exceeding the 10% effect dose could range from 0% to 1.9%, given the uncertainties that exist about how to parameterize the Monte Carlo exposure model. The corresponding probability of exceeding the 20% effects doses could range from 0% to 1.5%. Thus, small piscivorous mammals feeding in the UCR AOC are at a low risk of toxicity from exposure to mercury (Table I2-4).

The Monte Carlo analysis indicates that small piscivorous mammals have a 49.4% probability of total daily mercury intake exceeding the Appendix G benchmark of 0.0116 mg/kg bw/day. The lower and upper probability bounds of exposure have a 5% and 100% probability of exceeding the Appendix G benchmark, respectively. The probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I2-5.

#### ***Mercury in Middle Calcasieu River Area of Concern (MCR AOC)***

Integration of the methylmercury effects curve with the Monte Carlo curve for the exposure of the average-sized piscivorous mammals to mercury in the MCR AOC of the Calcasieu Estuary indicates that there is a 0% probability of exceeding the 10% and 20% effects doses (Table I2-4). The probability bounds analysis indicates that the probability of exceeding the 10% effect dose could range from 0% to 1.9%, given the uncertainties that exist about how to parameterize the Monte Carlo exposure model. The corresponding probability of exceeding the 20% effects doses could range from 0% to 1.5%. Thus, average-sized piscivorous mammals feeding in the MCR AOC are at a low risk of toxicity from exposure to mercury (Table I2-4).

The Monte Carlo analysis indicates that average-sized piscivorous mammals have a 38.5% probability of total daily mercury intake exceeding the Appendix G benchmark of 0.0116 mg/kg bw/day. The lower and upper probability bounds of exposure have a 4% and 99% probability of exceeding the Appendix G benchmark, respectively. The probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I2-5.

Integration of the methylmercury effects curve with the Monte Carlo curve for the exposure of the small piscivorous mammals to mercury in the MCR AOC of the Calcasieu Estuary indicates that there is a 0% probability of exceeding the 10% and 20% effects doses (Table I2-4). The probability bounds analysis indicates that the probability of exceeding the 10% effect dose could range from 0% to 2%, given the uncertainties that exist about how to parameterize the Monte Carlo exposure model. The corresponding probability of exceeding the 20% effects doses could range from 0% to 1.2%. Thus, small piscivorous mammals feeding in the MCR AOC are at a low risk of toxicity from exposure to mercury (Table I2-4).

The Monte Carlo analysis indicates that small piscivorous mammals have a 71% probability of total daily mercury intake exceeding the Appendix G benchmark of 0.0116 mg/kg bw/day. The lower and upper probability bounds of exposure have a 17% and 100% probability of exceeding the Appendix G benchmark, respectively. The probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I2-5.

### ***Mercury in Reference Areas***

Integration of the methylmercury effects curve with the Monte Carlo curve for the exposure of the average-sized piscivorous mammals to mercury in the reference areas of the Calcasieu Estuary indicates that there is a 0% probability of exceeding the 10%

and 20% effects doses (Table I2-4). The probability bounds analysis indicates that the probability of exceeding the 10% effect dose could range from 0% to 1%, given the uncertainties that exist about how to parameterize the Monte Carlo exposure model. The corresponding probability of exceeding the 20% effects doses is 0%. Thus, average-sized piscivorous mammals feeding in the reference areas of the Calcasieu Estuary are at a low risk of toxicity from exposure to mercury (Table I2-4). The Monte Carlo analysis indicates that average-sized piscivorous mammals have a 0% probability of total daily mercury intake exceeding the Appendix G benchmark of 0.0116 mg/kg bw/day. The lower and upper probability bounds of exposure have a 0% and 58% probability of exceeding the Appendix G benchmark, respectively. The probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I2-5.

Integration of the methylmercury effects curve with the Monte Carlo curve for the exposure of the small piscivorous mammals to mercury in the reference areas of the Calcasieu Estuary indicates that there is a 0% probability of exceeding the 10% and 20% effects doses (Table I2-4). The probability bounds analysis indicates that the probability of exceeding the 10% effect dose could range from 0% to 1.3%, given the uncertainties that exist about how to parameterize the Monte Carlo exposure model. The corresponding probability of exceeding the 20% effects doses is 0%. Thus, small piscivorous mammals feeding in the reference areas of the Calcasieu Estuary are at a low risk of toxicity from exposure to mercury (Table I2-4).

The Monte Carlo analysis indicates that small piscivorous mammals have a 4.1% probability of total daily mercury intake exceeding the Appendix G benchmark of 0.0116 mg/kg bw/day. The lower and upper probability bounds of exposure have a 0% and 81% probability of exceeding the Appendix G benchmark, respectively. The

probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I2-5.

***TCDD-TEQs in Bayou d'Inde Area of Concern (BI AOC)***

The effects metrics for TCDD-TEQs are based on the combined investigations conducted by Khera and Ruddick (1973) and Sparschu *et al.* (1971). The toxicological endpoint was fecundity of female rat. The dose-response curve indicated that 10% and 20% declines in fecundity would be expected at doses of 15.6 and 33.0 ng/kg bw/day, respectively.

Integration of the TCDD-TEQs effects curve with the Monte Carlo curve for the exposure of the average-sized piscivorous mammals to TCDD-TEQs in the BI AOC of the Calcasieu Estuary indicates that there is a 0% probability of exceeding the 10% and 20% effects doses (Table I2-4). The probability bounds analysis indicates that the probability of exceeding the 10% effect dose could range from 0% to 1.6%, given the uncertainties that exist about how to parameterize the Monte Carlo exposure model. The corresponding probability of exceeding the 20% effects doses is 0%. Figure I2-37 shows the estimated probabilities of effects of differing magnitude for average-sized piscivorous mammals in BI AOC. Thus, average-sized piscivorous mammals feeding in the BI AOC are at a low risk of toxicity from exposure to TCDD-TEQs (Table I2-4).

The Monte Carlo analysis indicates that average-sized piscivorous mammals have a 100% probability of total daily TCDD-TEQs intake exceeding the Appendix G benchmark of 1.52 ng/kg bw/day. The lower and upper probability bounds of exposure have a 98% and 100% probability of exceeding the Appendix G benchmark, respectively. The probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I2-5.

Integration of the TCDD-TEQs effects curve with the Monte Carlo curve for the exposure of the small piscivorous mammals to TCDD-TEQs in the BI AOC of the Calcasieu Estuary indicates that there is a 0% probability of exceeding the 10% and 20% effects doses (Table I2-4). The probability bounds analysis indicates that the probability of exceeding the 10% effect dose could range from 0% to 1.8%, given the uncertainties that exist about how to parameterize the Monte Carlo exposure model. The corresponding probability of exceeding the 20% effects doses is 0%. Figure I2-38 shows the estimated probabilities of effects of differing magnitude for small piscivorous mammals in BI AOC. Thus, small piscivorous mammals feeding in the BI AOC are at a low risk of toxicity from exposure to TCDD-TEQs (Table I2-4).

The Monte Carlo analysis indicates that small piscivorous mammals have a 100% probability of total daily TCDD-TEQs intake exceeding the Appendix G benchmark of 1.52 ng/kg bw/day. The lower and upper probability bounds of exposure have a 99% and 100% probability of exceeding the Appendix G benchmark, respectively. The probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I2-5.

#### ***TCDD-TEQs in Upper Calcasieu River Area of Concern (UCR AOC)***

Integration of the TCDD-TEQs effects curve with the Monte Carlo curve for the exposure of the average-sized piscivorous mammals to TCDD-TEQs in the UCR AOC of the Calcasieu Estuary indicates that there is a 0% probability of exceeding the 10% and 20% effects doses (Table I2-4). The probability bounds analysis indicates that the probability of exceeding the 10% and 20% effects doses is 0%, despite the uncertainties that exist about how to parameterize the Monte Carlo exposure model. Thus, average-sized piscivorous mammals feeding in the UCR AOC are at a low risk of toxicity from exposure to TCDD-TEQs (Table I2-4).

The Monte Carlo analysis indicates that average-sized piscivorous mammals have a 94.7% probability of total daily TCDD-TEQs intake exceeding the Appendix G benchmark of 1.52 ng/kg bw/day. The lower and upper probability bounds of exposure have a 17% and 100% probability of exceeding the Appendix G benchmark, respectively. The probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I2-5.

Integration of the TCDD-TEQs effects curve with the Monte Carlo curve for the exposure of the small piscivorous mammals to TCDD-TEQs in the UCR AOC of the Calcasieu Estuary indicates that there is a 0% probability of exceeding the 10% and 20% effects doses (Table I2-4). The probability bounds analysis indicates that the probability of exceeding the 10% and 20% effects doses is 0%, despite the uncertainties that exist about how to parameterize the Monte Carlo exposure model. Thus, small piscivorous mammals feeding in the UCR AOC are at a low risk of toxicity from exposure to TCDD-TEQs (Table I2-4).

The Monte Carlo analysis indicates that small piscivorous mammals have a 99.7% probability of total daily TCDD-TEQs intake exceeding the Appendix G benchmark of 1.52 ng/kg bw/day. The lower and upper probability bounds of exposure have a 34% and 100% probability of exceeding the Appendix G benchmark, respectively. The probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I2-5.

#### ***TCDD-TEQs in Middle Calcasieu River Area of Concern (MCR AOC)***

Integration of the TCDD-TEQs effects curve with the Monte Carlo curve for the exposure of the average-sized piscivorous mammals to TCDD-TEQs in the MCR AOC of the Calcasieu Estuary indicates that there is a 0% probability of exceeding the 10% and 20% effects doses (Table I2-4). The probability bounds analysis indicates that

the probability of exceeding the 10% and 20% effects doses is 0%, despite the uncertainties that exist about how to parameterize the Monte Carlo exposure model. Thus, average-sized piscivorous mammals feeding in the MCR AOC are at a low risk of toxicity from exposure to TCDD-TEQs (Table I2-4).

The Monte Carlo analysis indicates that average-sized piscivorous mammals have a 99% probability of total daily TCDD-TEQs intake exceeding the Appendix G benchmark of 1.52 ng/kg bw/day. The lower and upper probability bounds of exposure have a 0% and 100% probability of exceeding the Appendix G benchmark, respectively. The probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I2-5.

Integration of the TCDD-TEQs effects curve with the Monte Carlo curve for the exposure of the small piscivorous mammals to TCDD-TEQs in the MCR AOC of the Calcasieu Estuary indicates that there is a 0% probability of exceeding the 10% and 20% effects doses (Table I2-4). The probability bounds analysis indicates that the probability of exceeding the 10% and 20% effects doses is 0%, despite the uncertainties that exist about how to parameterize the Monte Carlo exposure model. Thus, small piscivorous mammals feeding in the MCR AOC are at a low risk of toxicity from exposure to TCDD-TEQs (Table I2-4).

The Monte Carlo analysis indicates that small piscivorous mammals have a 100% probability of total daily TCDD-TEQs intake exceeding the Appendix G benchmark of 1.52 ng/kg bw/day. The lower and upper probability bounds of exposure have a 0% and 100% probability of exceeding the Appendix G benchmark, respectively. The probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I2-5.

***TCDD-TEQs in Reference Areas***

Integration of the TCDD-TEQs effects curve with the Monte Carlo curve for the exposure of the average-sized piscivorous mammals to TCDD-TEQs in the reference areas of the Calcasieu Estuary indicates that there is a 0% probability of exceeding the 10% and 20% effects doses (Table I2-4). The probability bounds analysis indicates that the probability of exceeding the 10% and 20% effects doses is 0%, despite the uncertainties that exist about how to parameterize the Monte Carlo exposure model. Thus, average-sized piscivorous mammals feeding in the reference areas of the Calcasieu Estuary are at a low risk of toxicity from exposure to TCDD-TEQs (Table I2-4).

The Monte Carlo analysis indicates that average-sized piscivorous mammals have a 63.1% probability of total daily TCDD-TEQs intake exceeding the Appendix G benchmark of 1.52 ng/kg bw/day. The lower and upper probability bounds of exposure have a 9% and 100% probability of exceeding the Appendix G benchmark, respectively. The probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I2-5.

Integration of the TCDD-TEQs effects curve with the Monte Carlo curve for the exposure of the small piscivorous mammals to TCDD-TEQs in the reference areas of the Calcasieu Estuary indicates that there is a 0% probability of exceeding the 10% and 20% effects doses (Table I2-4). The probability bounds analysis indicates that the probability of exceeding the 10% and 20% effects doses is 0%, despite the uncertainties that exist about how to parameterize the Monte Carlo exposure model. Thus, small piscivorous mammals feeding in the reference areas of the Calcasieu Estuary are at a low risk of toxicity from exposure to TCDD-TEQs (Table I2-4).

The Monte Carlo analysis indicates that small piscivorous mammals have a 90.2% probability of total daily TCDD-TEQs intake exceeding the Appendix G benchmark of 1.52 ng/kg bw/day. The lower and upper probability bounds of exposure have a 29% and 100% probability of exceeding the Appendix G benchmark, respectively. The probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I2-5.

### ***Selenium in Bayou d'Inde Area of Concern (BI AOC)***

The effects metrics for selenium are based on the investigation conducted by Jacobs and Forst (1981). The toxicological endpoint was mortality of female rat. The dose-response curve indicated that 10% and 20% declines in mortality would be expected at doses of 1.08 and 1.35 mg/kg bw/day, respectively.

Integration of the selenium effects curve with the Monte Carlo curve for the exposure of the average-sized piscivorous mammals to selenium in the BI AOC of the Calcasieu Estuary indicates that there is a 0% probability of exceeding the 10% and 20% effects doses (Table I2-4). The probability bounds analysis indicates that the probability of exceeding the 10% effect dose could range from 0% to 2.5%, given the uncertainties that exist about how to parameterize the Monte Carlo exposure model. The corresponding probability of exceeding the 20% effects doses could range from 0% to 2%. Thus, average-sized piscivorous mammals feeding in the BI AOC are at a low risk of toxicity from exposure to selenium (Table I2-4).

The Monte Carlo analysis indicates that average-sized piscivorous mammals have a 31.8% probability of total daily selenium intake exceeding the Appendix G benchmark of 0.117 mg/kg bw/day. The lower and upper probability bounds of exposure have a 6% and 98% probability of exceeding the Appendix G benchmark,

respectively. The probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I2-5.

Integration of the selenium effects curve with the Monte Carlo curve for the exposure of the small piscivorous mammals to selenium in the BI AOC of the Calcasieu Estuary indicates that there is a 0% probability of exceeding the 10% and 20% effects doses (Table I2-4). The probability bounds analysis indicates that the probability of exceeding the 10% effect dose could range from 0% to 2.7%, given the uncertainties that exist about how to parameterize the Monte Carlo exposure model. The corresponding probability of exceeding the 20% effects doses could range from 0% to 2.4%. Thus, small piscivorous mammals feeding in the BI AOC are at a low risk of toxicity from exposure to selenium (Table I2-4).

The Monte Carlo analysis indicates that small piscivorous mammals have a 64.5% probability of total daily selenium intake exceeding the Appendix G benchmark of 0.117 mg/kg bw/day. The lower and upper probability bounds of exposure have 22% and 100% probability of exceeding the Appendix G benchmark, respectively. The probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I2-5.

#### ***Selenium in Middle Calcasieu River Area of Concern (MCR AOC)***

Integration of the selenium effects curve with the Monte Carlo curve for the exposure of the average-sized piscivorous mammals to selenium in the MCR AOC of the Calcasieu Estuary indicates that there is a 0% probability of exceeding the 10% and 20% effects doses (Table I2-4). The probability bounds analysis indicates that the probability of exceeding the 10% effect dose could range from 0% to 2.8%, given the uncertainties that exist about how to parameterize the Monte Carlo exposure model. The corresponding probability of exceeding the 20% effects doses could range from

0% to 2.5%. Thus, average-sized piscivorous mammals feeding in the MCR AOC are at a low risk of toxicity from exposure to selenium (Table I2-4).

The Monte Carlo analysis indicates that average-sized piscivorous mammals have a 37.8% probability of total daily selenium intake exceeding the Appendix G benchmark of 0.117 mg/kg bw/day. The lower and upper probability bounds of exposure have a 3% and 100% probability of exceeding the Appendix G benchmark, respectively. The probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I2-5.

Integration of the selenium effects curve with the Monte Carlo curve for the exposure of the small piscivorous mammals to selenium in the MCR AOC of the Calcasieu Estuary indicates that there is a 0% probability of exceeding the 10% and 20% effects doses (Table I2-4). The probability bounds analysis indicates that the probability of exceeding the 10% effect dose could range from 0% to 2.9%, given the uncertainties that exist about how to parameterize the Monte Carlo exposure model. The corresponding probability of exceeding the 20% effects doses could range from 0% to 2.6%. Thus, small piscivorous mammals feeding in the MCR AOC are at a low risk of toxicity from exposure to selenium (Table I2-4).

The Monte Carlo analysis indicates that small piscivorous mammals have a 70.8% probability of total daily selenium intake exceeding the Appendix G benchmark of 0.117 mg/kg bw/day. The lower and upper probability bounds of exposure have a 12% and 100% probability of exceeding the Appendix G benchmark, respectively. The probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I2-5.

### ***Selenium in Reference Areas***

Integration of the selenium effects curve with the Monte Carlo curve for the exposure of the average-sized piscivorous mammals to selenium in the reference areas of the Calcasieu Estuary indicates that there is a 0% probability of exceeding the 10% and 20% effects doses (Table I2-4). The probability bounds analysis indicates that the probability of exceeding the 10% effect dose could range from 0% to 2.5%, given the uncertainties that exist about how to parameterize the Monte Carlo exposure model. The corresponding probability of exceeding the 20% effects doses could range from 0% to 2.2%. Thus, average-sized piscivorous mammals feeding in the reference areas of the Calcasieu Estuary are at a low risk of toxicity from exposure to selenium (Table I2-4).

The Monte Carlo analysis indicates that average-sized piscivorous mammals have a 9.6% probability of total daily selenium intake exceeding the Appendix G benchmark of 0.117 mg/kg bw/day. The lower and upper probability bounds of exposure have a 0% and 91% probability of exceeding the Appendix G benchmark, respectively. The probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I2-5.

Integration of the selenium effects curve with the Monte Carlo curve for the exposure of the small piscivorous mammals to selenium in the reference areas of the Calcasieu Estuary indicates that there is a 0% probability of exceeding the 10% and 20% effects doses (Table I2-4). The probability bounds analysis indicates that the probability of exceeding the 10% effect dose could range from 0% to 2.7%, given the uncertainties that exist about how to parameterize the Monte Carlo exposure model. The corresponding probability of exceeding the 20% effects doses could range from 0% to 2.4%. Thus, small piscivorous mammals feeding in the reference areas of the Calcasieu Estuary are at a low risk of toxicity from exposure to selenium (Table I2-4).

The Monte Carlo analysis indicates that small piscivorous mammals have a 27.8% probability of total daily selenium intake exceeding the Appendix G benchmark of 0.117 mg/kg bw/day. The lower and upper probability bounds of exposure have a 0% and 100% probability of exceeding the Appendix G benchmark, respectively. The probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I2-5.

***Total PCBs in Bayou d'Inde Area of Concern (BI AOC)***

The effects metrics for total PCBs is based on the investigation conducted by Aulerich *et al.* (1985) and Aulerich and Ringer (1977). The toxicological endpoint was fecundity of female mink. The dose-response curve indicated that 10% and 20% declines in fecundity would be expected at doses of 0.0128 and 0.0272 mg/kg bw/day, respectively.

Integration of the PCBs effects curve with the Monte Carlo curve for the exposure of the average-sized piscivorous mammals to PCBs in the BI AOC of the Calcasieu Estuary indicates that there is a 100% probability of exceeding the 10% effect dose and a 83.9% probability of exceeding the 20% dose (Table I2-4). Further, there is a 50% probability of a 25% or greater reduction in reproductive fecundity and a 10% probability of a 38% or greater reduction in reproductive fecundity. The probability bounds analysis indicates that the probability of exceeding the 10% effect dose could range from 89% to 100%, given the uncertainties that exist about how to parameterize the Monte Carlo exposure model. The corresponding probability of exceeding the 20% effects doses could range from 14% to 100%. Figure I2-39 shows the estimated probabilities of effects of differing magnitude for average-sized piscivorous mammals in BI AOC. Thus, average-sized piscivorous mammals feeding in the BI AOC are at a high risk of toxicity from exposure to PCBs (Table I2-4).

The Monte Carlo analysis indicates that average-sized piscivorous mammals have a 100% probability of total daily PCBs intake exceeding the Appendix G benchmark of 0.00272 mg/kg bw/day. The lower and upper probability bounds of exposure have a 100% probability of exceeding the Appendix G benchmark, respectively. The probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I2-5.

Integration of the PCBs effects curve with the Monte Carlo curve for the exposure of the small piscivorous mammals to PCBs in the BI AOC of the Calcasieu Estuary indicates that there is a 100% probability of exceeding the 10% effect dose and a 97.7% probability of exceeding the 20% dose (Table I2-4). Further, there is a 50% probability of a 33% or greater increase in reproductive fecundity and a 10% probability of a 45% or greater increase in reproductive fecundity. The probability bounds analysis indicates that the probability of exceeding the 10% effect dose could range from 97% to 100%, given the uncertainties that exist about how to parameterize the Monte Carlo exposure model. The corresponding probability of exceeding the 20% effects doses could range from 36% to 100%. Figure I2-40 shows the estimated probabilities of effects of differing magnitude for small piscivorous mammals in BI AOC. Thus, small piscivorous mammals feeding in the BI AOC are at a high risk of toxicity from exposure to PCBs (Table I2-4).

The Monte Carlo analysis indicates that small piscivorous mammals have a 100% probability of total daily PCBs intake exceeding the Appendix G benchmark of 0.00272 mg/kg bw/day. The lower and upper probability bounds of exposure have a 100% probability of exceeding the Appendix G benchmark. The probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I2-5.

***PCBs in Reference Areas***

Integration of the PCBs effects curve with the Monte Carlo curve for the exposure of the average-sized piscivorous mammals to PCBs in the reference areas of the Calcasieu Estuary indicates that there is a 0% probability of exceeding the 10% and 20% effects doses (Table I2-4). There is, however, a 50% probability of a 4.5% or greater reduction in reproductive fecundity and a 10% probability of a 7% or greater reduction in reproductive fecundity. The probability bounds analysis indicates that the probability of exceeding the 10% effect dose could range from 0% to 68%, given the uncertainties that exist about how to parameterize the Monte Carlo exposure model. The corresponding probability of exceeding the 20% effects doses could range from 0% to 13%. Figure I2-41 shows the estimated probabilities of effects of differing magnitude for average-sized piscivorous mammals in the reference areas. Thus, average-sized piscivorous mammals feeding in the reference areas of the Calcasieu Estuary are at a low risk of toxicity from exposure to PCBs (Table I2-4).

The Monte Carlo analysis indicates that average-sized piscivorous mammals have a 99.8% probability of total daily PCBs intake exceeding the Appendix G benchmark of 0.00272 mg/kg bw/day. The lower and upper probability bounds of exposure have a 68% and 100% probability of exceeding the Appendix G benchmark, respectively. The probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I2-5.

Integration of the PCBs effects curve with the Monte Carlo curve for the exposure of the small piscivorous mammals to PCBs in the reference areas of the Calcasieu Estuary indicates that there is a 3.4% probability of exceeding the 10% effect dose, and a 0% probability of exceeding the 20% dose (Table I2-4). There is, however, a 50% probability of a 6% or greater reduction in reproductive fecundity and a 10% probability of a 9% or greater reduction in reproductive fecundity. The probability

bounds analysis indicates that the probability of exceeding the 10% effect dose could range from 0% to 88%, given the uncertainties that exist about how to parameterize the Monte Carlo exposure model. The corresponding probability of exceeding the 20% effects doses could range from 0% to 27%. Figure I2-42 shows the estimated probabilities of effects of differing magnitude for small piscivorous mammals in the reference areas. Thus, small piscivorous mammals feeding in the reference areas of the Calcasieu Estuary are at a low risk of toxicity from exposure to PCBs (Table I2-4).

The Monte Carlo analysis indicates that small piscivorous mammals have a 100% probability of total daily PCBs intake exceeding the Appendix G benchmark of 0.00272 mg/kg bw/day. The lower and upper probability bounds of exposure have a 86% and 100% probability of exceeding the Appendix G benchmark, respectively. The probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I2-5.

### ***Historical Data***

Levels of Aroclor 1254 in tissues of fish collected from CH2M Hill's Calcasieu Estuary Biological Monitoring Program were consistent with levels found in the Phase II Sampling Program. Levels in whole body determined in 2001 during Phase II Sampling and levels in fillet recorded since 1991 by CH2M Hill were used for statistical analysis. For comparison, fillet concentrations were estimated for the samples collected from the Phase II Sampling Program using the following equation:

$$C_f = C_{wb} / 2.3 \quad (3)$$

where  $C_f$  is concentration in fish fillet and  $C_{wb}$  is concentration in fish whole body (SAIC 1993). The equation is based on an empirical relationship described in Bevelhimer *et al.* (1996). Annual geometric mean concentrations in fillet of red

drum, black drum, spotted seatrout, sand seatrout and southern flounder were calculated for the three AOCs and the reference areas. The geometric mean concentration of Aroclor 1254 in fillet collected from the Upper Calcasieu River AOC during the Phase II Sampling Program was 0.013 mg/kg, with minimum and maximum concentrations of 0.002 mg/kg and 0.478 mg/kg, respectively. Since 1991, the annual geometric mean concentrations determined by CH2M Hill's Biological Monitoring Program ranged from 0.006 mg/kg to 0.040 mg/kg and the minimum and maximum concentrations were 0.005 mg/kg and 0.232 mg/kg, respectively (Figure I2-43).

The geometric mean concentration of Aroclor 1254 in fillet collected from the Bayou d'Inde AOC during the Phase II Sampling Program was 0.016 mg/kg, with minimum and maximum concentrations of 0.002 mg/kg and 0.230 mg/kg, respectively. Since 1991, the annual geometric mean concentrations determined by CH2M Hill's Biological Monitoring Program ranged from 0.028 mg/kg to 0.133 mg/kg and the minimum and maximum concentrations were 0.003 mg/kg and 1.080 mg/kg, respectively (Figure I2-44).

The geometric mean concentration of Aroclor 1254 in fillet collected from the Middle Calcasieu River AOC during the Phase II Sampling Program was 0.013 mg/kg, with a minimum and maximum concentration of 0.002 mg/kg and 0.317 mg/kg, respectively. Since 1991, the annual geometric mean concentrations determined by CH2M Hill's Biological Monitoring Program ranged from 0.008 mg/kg to 0.031 mg/kg and the minimum and maximum concentrations were 0.003 mg/kg and 0.221 mg/kg, respectively (Figure I2-45).

The geometric mean concentration of Aroclor 1254 in fillet collected from the Calcasieu Estuary reference areas during the Phase II Sampling Program was 0.006

mg/kg, with a minimum and maximum concentration of 0.002 mg/kg and 0.029 mg/kg, respectively. Since 1991, the annual geometric mean concentrations determined by CH2M Hill's Biological Monitoring Program ranged from 0.006 mg/kg to 0.016 mg/kg and the minimum and maximum concentrations were 0.003 mg/kg and 0.378 mg/kg, respectively (Figure I2-46).

The comparison of historical data sets between the Phase II Sampling Program and CH2M Hill's Biological Monitoring Program showed that there was less than one order of magnitude difference in levels of total PCBs in fish tissue between the ten years of historical data and data collected in the Phase II Sampling Program. In most cases, the difference was less than four fold. This demonstrates that the results of the ecological risk assessment for piscivorous mammals using data from the Phase II Sampling Program are likely to be temporally representative.

## **4.0 Uncertainty Analysis**

There are a number of sources of uncertainty in the assessments of risk to piscivorous mammals, including uncertainties in the conceptual model, in the exposure, effects, and risk assessments. As each of these sources of uncertainty can influence the estimations of risk, it is important to describe and, when possible, quantify the magnitude and direction of such uncertainties. In this way, it is possible to evaluate the level of confidence that can be placed in the assessments conducted. The uncertainties associated with the assessment of risks to piscivorous mammals are described in the following sections.

**Uncertainties Associated with the Conceptual Model** - The conceptual model is intended to define the linkages between stressors, potential exposure, and predicted

effects on ecological receptors. As such, the conceptual model provides the scientific basis for selecting assessment and measurement endpoints to support the risk assessment process. Potential uncertainties arise from lack of knowledge regarding ecosystem functions, failure to adequately address spatial and temporal variability in the evaluations of sources, fate, and effects, omission of stressors, and overlooking secondary effects (USEPA 1998). The types of uncertainties associated with the conceptual model that links contaminant sources to effects on piscivorous mammals include those associated with the identification of COCs, environmental fate and transport of COCs, exposure pathways, receptors at risk, and ecological effects. Of these, the identification of exposure pathways probably represents the primary source of uncertainty in the conceptual model. In this assessment, it was assumed that exposure to contaminated food represents the most important pathway for exposing piscivorous mammals to COCs. Although unlikely to be important, other pathways could contribute to exposure and perhaps increase risk somewhat.

**Uncertainties Associated with the Exposure Assessment** - The exposure assessment is intended to describe the actual or potential co-occurrence of stressors with receptors. As such, the exposure assessment identifies the exposure pathways and the intensity and extent of contact with stressors for each receptor or group of receptors at risk. There are a number of potential sources of uncertainty in the exposure assessment, including measurement errors, extrapolation errors, and data gaps.

In this assessment, chemical analyses of tissue residues in fish and invertebrates were used to evaluate exposure of piscivorous mammals to COCs. Analytical errors and descriptive errors represent potential sources of uncertainty. Three approaches were used to address concerns relative to these sources of uncertainty.

First, analytical errors were evaluated using information on the accuracy, precision, and detection limits (DL) generated to support the Phase I and Phase II sampling programs. The results of this analysis indicated that most of the data used in this assessment met the project data quality objectives (see Appendix B1 for more details). Second, all data entry, data translation, and data manipulations were audited to ensure their accuracy. Data auditing involved 10% number-for-number checks against the primary data source initially, increasing to 100% number-for-number checks if significant errors were detected in the initial auditing step. Finally, statistical analyses of data were conducted to evaluate data distributions, identify appropriate summary statistics, and evaluate variability in the observations. Using these techniques, we were able to identify outliers and, if the outliers were due to an error, correct the outlier values.

According to the Monte Carlo sensitivity analyses, the FMR slope and power term were among the most influential variables driving the predicted intake rates. Unfortunately, a precise estimate of the FMR was not possible as suitable measured metabolic rates for piscivorous mammals were not available in the literature. Instead, the FMR for piscivorous mammals was estimated using allometric equations. This introduced some degree of uncertainty into the exposure estimates because the allometric relationships were not only associated with some fitting error, but also were based on many mammal species, some of which were very different from those represented here. However, given the lack of empirical data on species specific to the current assessment, it is difficult to judge the magnitude of the uncertainty introduced by the use of the allometric model rather than the empirical data.

Other sensitive variables that influenced the exposure estimates included the gross energy of food and the food assimilation efficiency. These variables also were somewhat uncertain because no feeding studies were specifically performed in the

Calcasieu Estuary on the species of interest. Rather, diet compositions were matched to those reported in the literature from other geographical locations. As a consequence, the quantification of food gross energy and assimilation efficiency was limited to the fish food group, without considering specific fish species. Furthermore, the estimates were uncertain, because they were approximated using gross energy and assimilation efficiency data for generic fish. Prey tissue sample sizes were small for many of the COC analyses in the AOCs, thus adding to the uncertainty in the piscivorous mammal exposure characterization.

**Uncertainties in the Effects Assessment** - The effects assessment is intended to describe the effects caused by stressors, link them to the assessment endpoints, and evaluate how effects change with fluctuations in the levels (i.e., concentrations or doses) of the various stressors. There are several sources of uncertainty in the assessment of effects including measurement errors, extrapolation errors, model fit errors, and data gaps.

The greatest source of uncertainty for the effects characterization is the lack of toxicity studies in which the representative species were dosed with TEQs and selenium. There were no toxicity studies available that treated mink, river otters, or another suitable wild piscivorous mammal, with doses of these COCs. Studies involving surrogate species, namely rats, were used instead. This added another degree of uncertainty because it is not known whether laboratory raised and tested animals have the same sensitivity as those living in the wild. Studies of the reproductive success of piscivorous mammals performed *in situ* in the Calcasieu Estuary were also not available. Such site specific field studies would have been more able to account for the specific characteristics of the Calcasieu Estuary ecosystem.

Another significant source of uncertainty in the risk assessment for piscivorous mammals is the lack of information available on abundance of piscivorous mammals across chemical gradients in the Calcasieu Estuary, and lack of toxicity studies on the responses of piscivorous mammals fed prey collected from the estuary.

## 5.0 Conclusions

The risk characterization results indicate that there is a low probability that exposure to mercury, TCDD-TEQs, and selenium will cause significant adverse effects to piscivorous mammals foraging in the Calcasieu Estuary. This statement holds true even when account is taken of the smallest animals in the guild. The analyses indicate that piscivorous mammals of any size are at high risk from exposure to total PCBs in BI AOC.

There are several limitations of the probabilistic risk analyses that influence our confidence regarding the above risk statements. These include:

- The sensitivity analyses for the Monte Carlo simulations indicated that the most important input variables were the slope and power terms used to estimate free metabolic rate (*FMR*). The *FMR* used in the analyses was based on the allometric equation from Nagy (1987). No *in situ* measurements of this variable are available for piscivorous mammals. The potential magnitude and direction of the uncertainty associated with lack of empirical data on metabolic rate are unknown. We did, however, investigate the possible consequences of the uncertainty in this variable due to model error (i.e., the error associated with the lack of fit of the

allometric model that relates *FMR* to body weight) in the probability bounds analysis;

- Sample size for COCs in fish and invertebrate tissues was generally limited. Although we accounted for this source of uncertainty in our analyses, it is possible that additional data would substantially change the distributions for the tissue residue variables (particularly if the samples were biased toward relatively contaminated or uncontaminated areas); and,
- The effects analyses pointed out several key sources of uncertainty. First, no data were available for any wild piscivorous mammalian species. Second, differing environmental conditions between the laboratory and the field introduces uncertainty to the estimation of effects doses.

The above described limitations are common to wildlife risk assessments and indicate the value of having other lines of evidence to help characterize risks. Biological surveys and ambient toxicity testing are two such lines of evidence. No *in situ* or whole-media feeding studies are available, however, for piscivorous mammals in the Calcasieu estuary. Formal biological surveys that relate degree of COC contamination to abundances of different piscivorous mammalian species have not been conducted. While the evidence presented certainly cannot be used to rule out the possibility that COCs are causing adverse effects to piscivorous mammals in the Calcasieu Estuary, it does seem unlikely that COCs are causing widespread impacts except for total PCBs which posed a high risk in the BI AOC.

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# Tables

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**Table I2-1. Results of deterministic risk assessment for piscivorous mammals in the Calcasieu Estuary.**

Contaminant of Concern (COC)	Area	Risk Quotient	Proceed to Probabilistic Assessment?
<i>Mercury</i>	Bayou d'Inde	12.2	Yes
	Upper Calcasieu River	3.3	Yes
	Middle Calcasieu River	3.9	Yes
	Reference Areas	2.3	Yes
<i>TCDD-TEQs</i>	Bayou d'Inde	22.7	Yes
	Upper Calcasieu River	7.5	Yes
	Middle Calcasieu River	10.8	Yes
	Reference Areas	1.7	Yes
<i>Selenium</i>	Bayou d'Inde	2.2	Yes
	Upper Calcasieu River	1.9	No
	Middle Calcasieu River	2.5	Yes
	Reference Areas	1.8	Yes
<i>Total PCBs</i>	Bayou d'Inde	125	Yes
	Upper Calcasieu River	46	No
	Middle Calcasieu River	40.8	No
	Reference Areas	49.8	Yes

TCDD = tetrachlorodibenzo-*p*-dioxin; TEQs = toxic equivalents; PCBs = polychlorinated biphenyls.

**Table I2-2. Monte Carlo input variables.**

Variable	Distribution	Parameters		
Body weight (BW) - average-sized species (g)	normal	Mean = 3,960; SD = 396		
Body weight (BW) - small species (g)	normal	Mean = 608; SD =66.9		
Free Metabolic Rate - average-sized and small species (FMR; Kcal/kg bw/day)	FMR = aBW <sup>b</sup>			
a = FMR-slope	normal	Mean = 0.412 ; SD = 0.058		
b = FMR-power	normal	Mean = 0.862 ; SD = 0.026		
Assimilation Efficiency - Fish (AE <sub>f</sub> , unitless)	beta	á = 65; â = 6.7; scale =1		
Assimilation Efficiency - Invertebrates (AE <sub>i</sub> , unitless)	beta	á = 65; â = 10; scale =1		
Gross Energy - Fish (GE <sub>f</sub> ; Kcal/kg)	normal	Mean = 1200; SD = 240		
Gross Energy - Invertebrates (GE <sub>i</sub> ; Kcal/kg)	normal	Mean = 1050; SD = 225		
<b>Contaminants of Concern (COCs)</b>				
COC	Area	Tissue Classification		
Mercury	Bayou d'Inde	C <sub>invert 1A,B-2A</sub> (mg/kg ww)	lognormal	Mean = 0.0374; SD = 0.000357
		C <sub>fish 1-2A,B</sub> (mg/kg ww)	lognormal	Mean = 0.188; SD = 0.00536
		C <sub>fish 3A,B</sub> (mg/kg ww)	lognormal	Mean = 0.133; SD = 0.00174
		C <sub>fish 4A,B</sub> (mg/kg ww)	lognormal	Mean = 0.168; SD = 0.00350
	Upper Calcasieu River	C <sub>invert 1A,B-2A</sub> (mg/kg ww)	lognormal	Mean = 0.0197; SD = 0.000408
		C <sub>fish 1-2A,B</sub> (mg/kg ww)	lognormal	Mean = 0.0445; SD = 0.000995
		C <sub>fish 3A,B</sub> (mg/kg ww)	lognormal	Mean = 0.0607; SD = 0.00191
		C <sub>fish 4A,B</sub> (mg/kg ww)	lognormal	Mean = 0.0761; SD = 0.00255
	Middle Calcasieu River	C <sub>invert 2A</sub> (mg/kg ww)	lognormal	Mean = 0.0241; SD = 0.000153
		C <sub>fish 1-2A,B</sub> (mg/kg ww)	lognormal	Mean = 0.0589; SD = 0.000791
		C <sub>fish 3A,B</sub> (mg/kg ww)	lognormal	Mean = 0.0524; SD = 0.000689
		C <sub>fish 4A,B</sub> (mg/kg ww)	lognormal	Mean = 0.0873; SD = 0.00235
	Reference Areas	C <sub>invert 2A</sub> (mg/kg ww)	lognormal	Mean = 0.00750; SD = 0.0000536
		C <sub>fish 1-2A,B</sub> (mg/kg ww)	lognormal	Mean = 0.0244; SD = 0.000255
		C <sub>fish 3A,B</sub> (mg/kg ww)	lognormal	Mean = 0.0268; SD = 0.000510
		C <sub>fish 4A,B</sub> (mg/kg ww)	lognormal	Mean = 0.0698; SD = 0.00291

**Table I2-2. Monte Carlo input variables.**

Variable			Distribution	Parameters
COC (cont.)	Area (cont.)	Tissue Classification (cont.)		
TCDD-TEQs	Bayou d'Inde	$C_{\text{invert } 2A}$ (ng/kg ww)	lognormal	Mean = 22.3; SD = 0.462
		$C_{\text{fish } 1-2A,B}$ (ng/kg ww)	lognormal	Mean = 29.6; SD = 0.141
		$C_{\text{fish } 3A,B}$ (ng/kg ww)	lognormal	Mean = 54.5; SD = 1.90
		$C_{\text{fish } 4A,B}$ (ng/kg ww)	lognormal	Mean = 87.9 ; SD = 6.68
	Upper Calcasieu River	$C_{\text{invert } 2A}$ (ng/kg ww)	lognormal	Mean = 5.26; SD = 0.0834
		$C_{\text{fish } 1-2A,B}$ (ng/kg ww)	lognormal	Mean = 7.49; SD = 0.215
		$C_{\text{fish } 3A,B}$ (ng/kg ww)	lognormal	Mean = 29.9; SD = 0.884
		$C_{\text{fish } 4A,B}$ (ng/kg ww)	lognormal	Mean = 25.5; SD = 1.36
	Middle Calcasieu River	$C_{\text{invert } 2A}$ (ng/kg ww)	lognormal	Mean = 7.09; SD = 0.0178
		$C_{\text{fish } 1-2A,B}$ (ng/kg ww)	lognormal	Mean = 16.3; SD = 0.482
		$C_{\text{fish } 3A,B}$ (ng/kg ww)	lognormal	Mean = 14.3; SD = 0.561
		$C_{\text{fish } 4A,B}$ (ng/kg ww)	lognormal	Mean = 31.1; SD = 1.68
	Reference Areas	$C_{\text{invert } 2A}$ (ng/kg ww)	lognormal	Mean = 0; SD = 0
		$C_{\text{fish } 1-2A,B}$ (ng/kg ww)	lognormal	Mean = 7.64; SD = 0.0934
		$C_{\text{fish } 3A,B}$ (ng/kg ww)	lognormal	Mean = 21.4; SD = 0
		$C_{\text{fish } 4A,B}$ (ng/kg ww)	lognormal	Mean = 3.90 ; SD = 0.0862
Selenium	Bayou d'Inde	$C_{\text{invert } 1A,B-2A}$ (mg/kg ww)	lognormal	Mean = 0.457; SD = 0.00128
		$C_{\text{fish } 1-2A,B}$ (mg/kg ww)	lognormal	Mean = 0.563; SD = 0.00612
		$C_{\text{fish } 3A,B}$ (mg/kg ww)	lognormal	Mean = 0.526; SD = 0.00482
		$C_{\text{fish } 4A,B}$ (mg/kg ww)	lognormal	Mean = 0.480; SD = 0.00434
	Middle Calcasieu River	$C_{\text{invert } 2A}$ (mg/kg ww)	lognormal	Mean = 0.463; SD = 0.00533
		$C_{\text{fish } 1-2A,B}$ (mg/kg ww)	lognormal	Mean = 0.498; SD = 0.0146
		$C_{\text{fish } 3A,B}$ (mg/kg ww)	lognormal	Mean = 0.759; SD = 0.0204
		$C_{\text{fish } 4A,B}$ (mg/kg ww)	lognormal	Mean = 0.697; SD = 0.00612
	Reference Areas	$C_{\text{invert } 2A}$ (mg/kg ww)	lognormal	Mean = 0.422; SD = 0.00582
		$C_{\text{fish } 1-2A,B}$ (mg/kg ww)	lognormal	Mean = 0.304; SD = 0.00543
		$C_{\text{fish } 3A,B}$ (mg/kg ww)	lognormal	Mean = 0.678; SD = 0.0136
		$C_{\text{fish } 4A,B}$ (mg/kg ww)	lognormal	Mean = 0.464; SD = 0.00773

**Table I2-2. Monte Carlo input variables.**

Variable			Distribution	Parameters
COC (cont.)	Area (cont.)	Tissue Classification (cont.)		
Total PCBs	Bayou D'Inde	$C_{\text{invert } 1A,B-2A}$ (mg/kg ww)	lognormal	Mean = 0.0354; SD = 0.000969
		$C_{\text{fish } 1-2A,B}$ (mg/kg ww)	lognormal	Mean = 0.113; SD = 0.00513
		$C_{\text{fish } 3A,B}$ (mg/kg ww)	lognormal	Mean = 0.362; SD = 0.0248
		$C_{\text{fish } 4A,B}$ (mg/kg ww)	lognormal	Mean = 0.602; SD = 0.0737
	Reference Areas	$C_{\text{invert } 2A}$ (mg/kg ww)	lognormal	Mean = 0.0104; SD = 0.000281
		$C_{\text{fish } 1-2A,B}$ (mg/kg ww)	lognormal	Mean = 0.0336; SD = 0.00156
		$C_{\text{fish } 3A,B}$ (mg/kg ww)	lognormal	Mean = 0.0290; SD = 0.00105
		$C_{\text{fish } 4A,B}$ (mg/kg ww)	lognormal	Mean = 0.0315; SD = 0.00153

TCDD = tetrachlorodibenzo-*p* -dioxin; TEQs = toxic equivalents; PCBs = polychlorinated biphenyls.

**Table I2-3. Probability bounds input variables.**

Variable	Distribution	Parameters		
Body weight (BW) - average-sized species (g)	normal	Mean = 3,960; SD = 396.1		
Body weight (BW) - small species (g)	normal	Mean = 608; SD =66.9		
Free Metabolic Rate - average-sized and small species (FMR; Kcal/kg bw/day)	FMR = aBW <sup>b</sup>			
a = FMR-slope	normal	Mean = 0.412 ; SD = 0.058		
b = FMR-power	normal	Mean = 0.862 ; SD = 0.026		
Assimilation Efficiency - Fish (AE <sub>f</sub> , unitless)	minmaxmean	0.77, 0.98, 0.91		
Assimilation Efficiency - Invertebrates (AE <sub>i</sub> , unitless)	minmaxmean	0.72, 0.96, 0.87		
Gross Energy - Fish (GE <sub>f</sub> ; Kcal/kg)	normal	Mean = 1200; SD =240		
Gross Energy - Invertebrates (GE <sub>i</sub> ; Kcal/kg)	normal	Mean = 1050; SD =225		
<b>Contaminants of Concern (COCs)</b>				
COC	Area	Tissue Classification		
Mercury	Bayou d'Inde	C <sub>invert 1A,B-2A</sub> (mg/kg ww)	lognormal	Mean = 0.0438; SD = 0.00695
		C <sub>fish 1-2A,B</sub> (mg/kg ww)	lognormal	Mean = 0.196; SD = 0.0213
		C <sub>fish 3A,B</sub> (mg/kg ww)	lognormal	Mean = 0.150; SD = 0.0192
		C <sub>fish 4A,B</sub> (mg/kg ww)	lognormal	Mean = 0.188; SD = 0.0329
	Upper Calcasieu River	C <sub>invert 1A,B-2A</sub> (mg/kg ww)	lognormal	Mean = 0.0221; SD = 0.00355
		C <sub>fish 1-2A,B</sub> (mg/kg ww)	lognormal	Mean = 0.0485; SD = 0.00621
		C <sub>fish 3A,B</sub> (mg/kg ww)	lognormal	Mean = 0.0674; SD = 0.0128
		C <sub>fish 4A,B</sub> (mg/kg ww)	lognormal	Mean = 0.0827; SD = 0.0140
	Middle Calcasieu River	C <sub>invert 2A</sub> (mg/kg ww)	lognormal	Mean = 0.0289; SD = 0.00421
		C <sub>fish 1-2A,B</sub> (mg/kg ww)	lognormal	Mean = 0.0666; SD = 0.00973
		C <sub>fish 3A,B</sub> (mg/kg ww)	lognormal	Mean = 0.0597; SD = 0.00930
		C <sub>fish 4A,B</sub> (mg/kg ww)	lognormal	Mean = 0.106; SD = 0.0286
	Reference Areas	C <sub>invert 2A</sub> (mg/kg ww)	lognormal	Mean = 0.00999; SD = 0.00238
		C <sub>fish 1-2A,B</sub> (mg/kg ww)	lognormal	Mean = 0.0292; SD = 0.00559
		C <sub>fish 3A,B</sub> (mg/kg ww)	lognormal	Mean = 0.0301; SD = 0.00512
		C <sub>fish 4A,B</sub> (mg/kg ww)	lognormal	Mean = 0.0770; SD = 0.0187

**Table I2-3. Probability bounds input variables.**

Variable			Distribution	Parameters
COC (cont.)	Area (cont.)	Tissue Classification (cont.)		
TCDD-TEQs	Bayou d'Inde	$C_{\text{invert } 2A}$ (ng/kg ww)	uniform	Min = 11.9; Max = 43.0
		$C_{\text{fish } 1-2A,B}$ (ng/kg ww)	lognormal	Mean = 34.2; SD = 5.92
		$C_{\text{fish } 3A,B}$ (ng/kg ww)	lognormal	Mean = 91.8; SD = 54.6
		$C_{\text{fish } 4A,B}$ (ng/kg ww)	uniform	Min = 3.49; Max = 258
	Upper Calcasieu River	$C_{\text{invert } 2A}$ (ng/kg ww)	uniform	Min = 2.40; Max = 6.96
		$C_{\text{fish } 1-2A,B}$ (ng/kg ww)	lognormal	Mean = 12.5; SD = 6.90
		$C_{\text{fish } 3A,B}$ (ng/kg ww)	uniform	Min = 7.76; Max = 85.7
		$C_{\text{fish } 4A,B}$ (ng/kg ww)	uniform	Min = 5.13; Max = 28.4
	Middle Calcasieu River	$C_{\text{invert } 2A}$ (ng/kg ww)	uniform	Min = 6.28; Max = 7.66
		$C_{\text{fish } 1-2A,B}$ (ng/kg ww)	uniform	Mean = 2.92; SD = 23.1
		$C_{\text{fish } 3A,B}$ (ng/kg ww)	uniform	Min = 3.61; Max = 23.4
		$C_{\text{fish } 4A,B}$ (ng/kg ww)	uniform	Min = 3.80; Max = 128
	Reference Areas	$C_{\text{invert } 2A}$ (ng/kg ww)	lognormal	Mean = 0; SD = 0
		$C_{\text{fish } 1-2A,B}$ (ng/kg ww)	uniform	Min = 3.99; Max = 9.88
		$C_{\text{fish } 3A,B}$ (ng/kg ww)	lognormal	Mean = 21.4; SD = 0
		$C_{\text{fish } 4A,B}$ (ng/kg ww)	uniform	Min = 1.54; Max = 8.70
Selenium	Bayou d'Inde	$C_{\text{invert } 1A,B-2A}$ (mg/kg ww)	lognormal	Mean = 0.759; SD = 0.126
		$C_{\text{fish } 1-2A,B}$ (mg/kg ww)	lognormal	Mean = 0.599; SD = 0.0418
		$C_{\text{fish } 3A,B}$ (mg/kg ww)	lognormal	Mean = 0.593; SD = 0.0698
		$C_{\text{fish } 4A,B}$ (mg/kg ww)	lognormal	Mean = 0.554; SD = 0.0674
	Middle Calcasieu River	$C_{\text{invert } 2A}$ (mg/kg ww)	lognormal	Mean = 0.548; SD = 0.0963
		$C_{\text{fish } 1-2A,B}$ (mg/kg ww)	lognormal	Mean = 0.560; SD = 0.116
		$C_{\text{fish } 3A,B}$ (mg/kg ww)	lognormal	Mean = 0.856; SD = 0.179
		$C_{\text{fish } 4A,B}$ (mg/kg ww)	lognormal	Mean = 0.843; SD = 0.150
	Reference Areas	$C_{\text{invert } 2A}$ (mg/kg ww)	lognormal	Mean = 0.567; SD = 0.167
		$C_{\text{fish } 1-2A,B}$ (mg/kg ww)	lognormal	Mean = 0.363; SD = 0.0818
		$C_{\text{fish } 3A,B}$ (mg/kg ww)	lognormal	Mean = 0.771; SD = 0.142
		$C_{\text{fish } 4A,B}$ (mg/kg ww)	lognormal	Mean = 0.520; SD = 0.0799

**Table I2-3. Probability bounds input variables.**

Variable			Distribution	Parameters
COC (cont.)	Area (cont.)	Tissue Classification (cont.)		
Total PCBs	Bayou D'Inde	$C_{\text{invert } 1A,B-2A}$ (mg/kg ww)	lognormal	Mean = 0.0403; SD = 0.00878
		$C_{\text{fish } 1-2A,B}$ (mg/kg ww)	lognormal	Mean = 0.117; SD = 0.0156
		$C_{\text{fish } 3A,B}$ (mg/kg ww)	lognormal	Mean = 0.394; SD = 0.118
		$C_{\text{fish } 4A,B}$ (mg/kg ww)	lognormal	Mean = 0.715; SD = 0.333
	Reference Areas	$C_{\text{invert } 2A}$ (mg/kg ww)	lognormal	Mean = 0.0131; SD = 0.00483
		$C_{\text{fish } 1-2A,B}$ (mg/kg ww)	lognormal	Mean = 0.0422; SD = 0.0173
		$C_{\text{fish } 3A,B}$ (mg/kg ww)	lognormal	Mean = 0.0328; SD = 0.00786
		$C_{\text{fish } 4A,B}$ (mg/kg ww)	lognormal	Mean = 0.0349; SD = 0.00905

TCDD = tetrachlorodibenzo-*p*-dioxin; TEQs = toxic equivalents; PCBs = polychlorinated biphenyls.

**Table I2-4. Summary of exceedance probabilities for piscivorous mammals from Calcasieu Estuary.**

Area/Contaminant of Concern (COC)	Probability of Exceedance (%)											
	Average-Sized Piscivorous Mammals						Small Piscivorous Mammals					
	LPB		FOMC		UPB		LPB		FOMC		UPB	
	10%	20%	10%	20%	10%	20%	10%	20%	10%	20%	10%	20%
<b><i>Mercury</i></b>												
Bayou D'Inde	0	0	0	0	12	7	0	0	0	0	26	14
Upper Calcasieu River	0	0	0	0	0	0	0	0	0	0	1.9	1.5
Middle Calcasieu River	0	0	0	0	1.9	1.5	0	0	0	0	2	1.2
Reference	0	0	0	0	1	0	0	0	0	0	1.3	0
<b><i>TCDD-TEQs</i></b>												
Bayou D'Inde	0	0	0	0	1.6	0	0	0	0	0	1.8	0
Upper Calcasieu River	0	0	0	0	0	0	0	0	0	0	0	0
Middle Calcasieu River	0	0	0	0	0	0	0	0	0	0	0	0
Reference Areas	0	0	0	0	0	0	0	0	0	0	0	0
<b><i>Selenium</i></b>												
Bayou D'Inde	0	0	0	0	2.5	2	0	0	0	0	2.7	2.4
Middle Calcasieu River	0	0	0	0	2.8	2.5	0	0	0	0	2.9	2.6
Reference Areas	0	0	0	0	2.5	2.2	0	0	0	0	2.7	2.4
<b><i>Total PCBs</i></b>												
Bayou d'Inde	89	14	100	83.9	100	100	97	36	100	97.7	100	100
Reference Areas	0	0	0	0	68	13	0	0	3.4	0	88	27

LPB = Lower Probability Bound; FOMC = First Order Monte Carlo; UPB = Upper Probability Bound; TCDD = tetrachlorodibenzo-*p*-dioxin; TEQs = toxic equivalents; PCBs = polychlorinated biphenyls.

**Table I2-5. Probabilities of exposure of piscivorous mammals to contaminants of concern (COCs) exceeding Appendix G ecological risk assessment benchmarks in the Calcasieu Estuary system.**

Area/Contaminant of Concern (COC)	Benchmark	Probability of Exposure Exceeding Appendix G Benchmark (%)					
		Average-Sized Piscivorous Mammal			Small Piscivorous Mammal		
		LPB	FOMC	UPB	LPB	FOMC	UPB
<b>Mercury</b>							
Bayou D’Inde	0.0116 mg/kg bw/d	95	100	100	99	100	100
Upper Calcasieu River		0	21.9	93	5	49.4	100
Middle Calcasieu River		4	38.5	99	17	71	100
Reference Areas		0	0	58	0	4.1	81
<b>TCDD-TEQs</b>							
Bayou D’Inde	1.52 ng/kg bw/d	98	100	100	99	100	100
Upper Calcasieu River		17	94.7	100	34	99.7	100
Middle Calcasieu River		0	100	100	0	99	100
Reference Areas		9	63.1	100	29	90.2	100
<b>Selenium</b>							
Bayou D’Inde	0.117 mg/kg bw/d	6	31.8	98	22	64.5	100
Middle Calcasieu River		3	37.8	100	12	70.8	100
Reference Areas		0	9.6	91	0	27.8	100
<b>Total PCBs</b>							
Bayou D’Inde	0.00272 mg/kg bw/d	100	100	100	100	100	100
Reference Areas		68	99.8	100	86	100	100

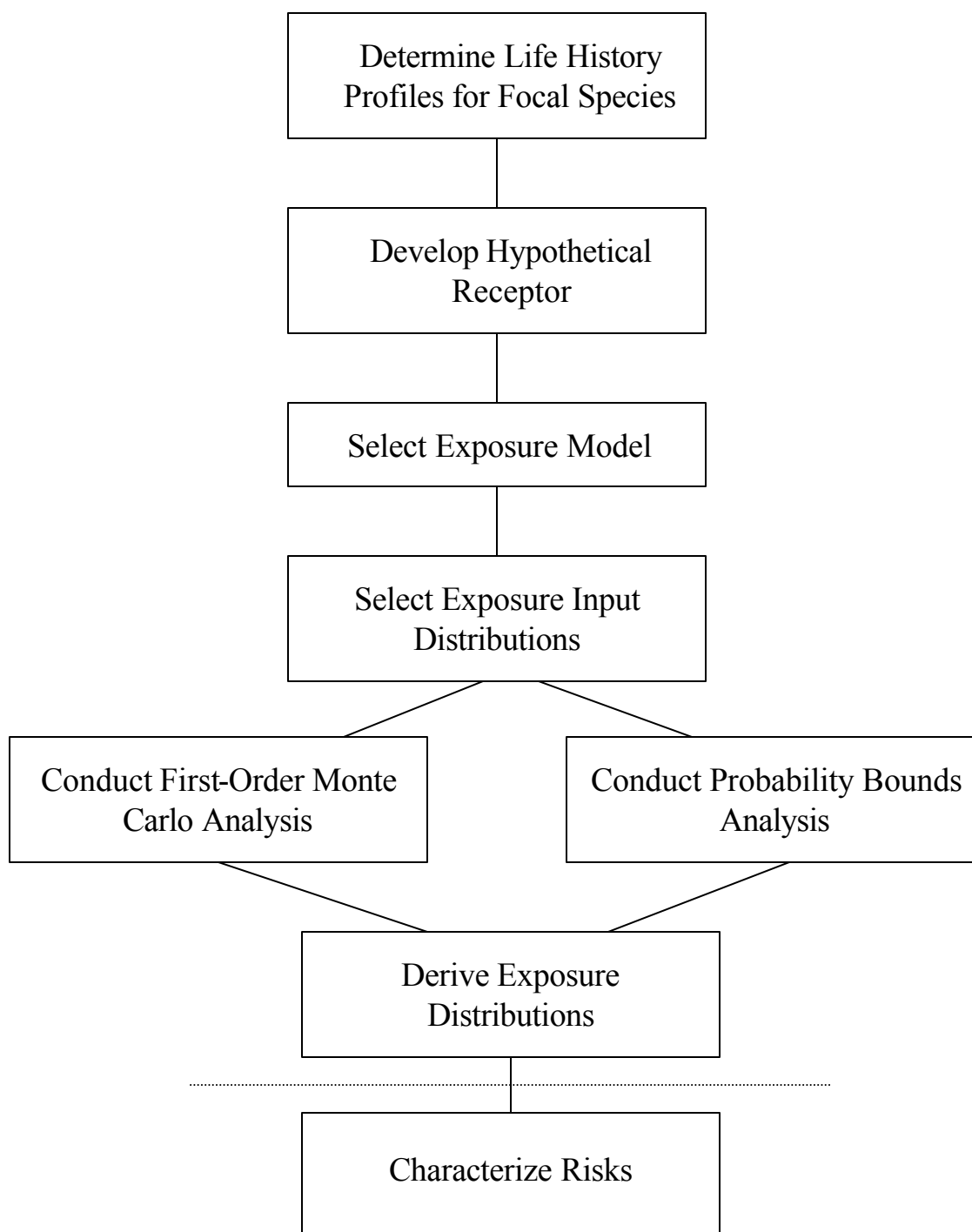
LPB = Lower Probability Bound; FOMC = First Order Monte Carlo; UPB = Upper Probability Bound; TCDD = tetrachlorodibenzo-*p*-dioxin; TEQs = toxic equivalents; PCBs = polychlorinated biphenyls.

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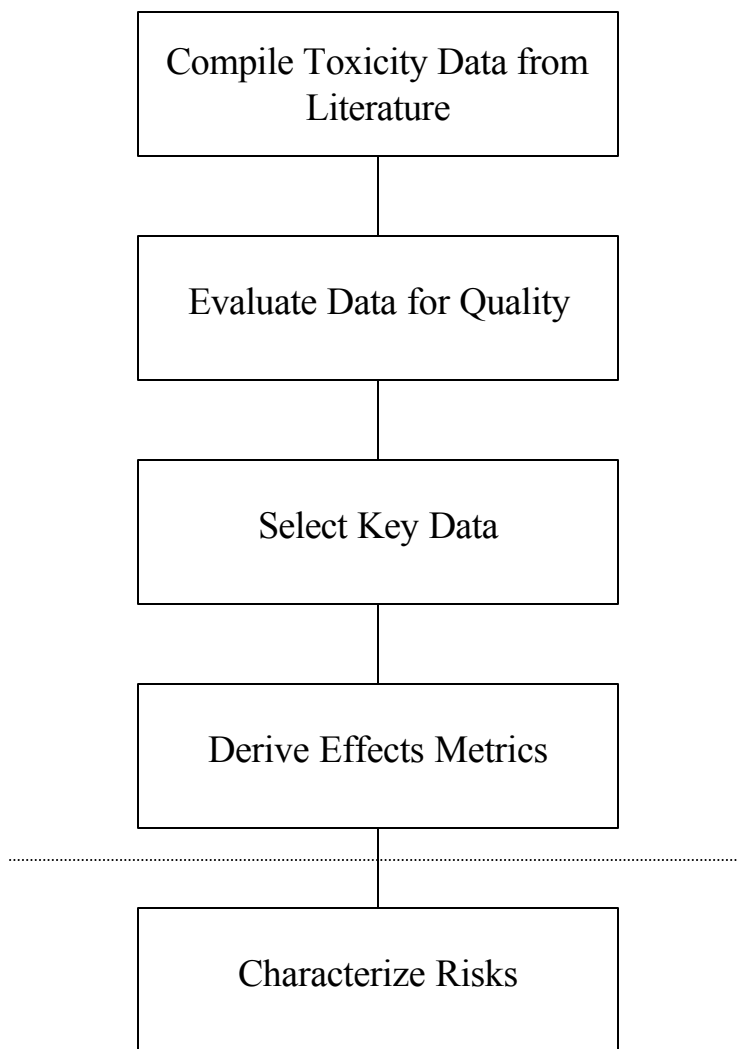
# Figures

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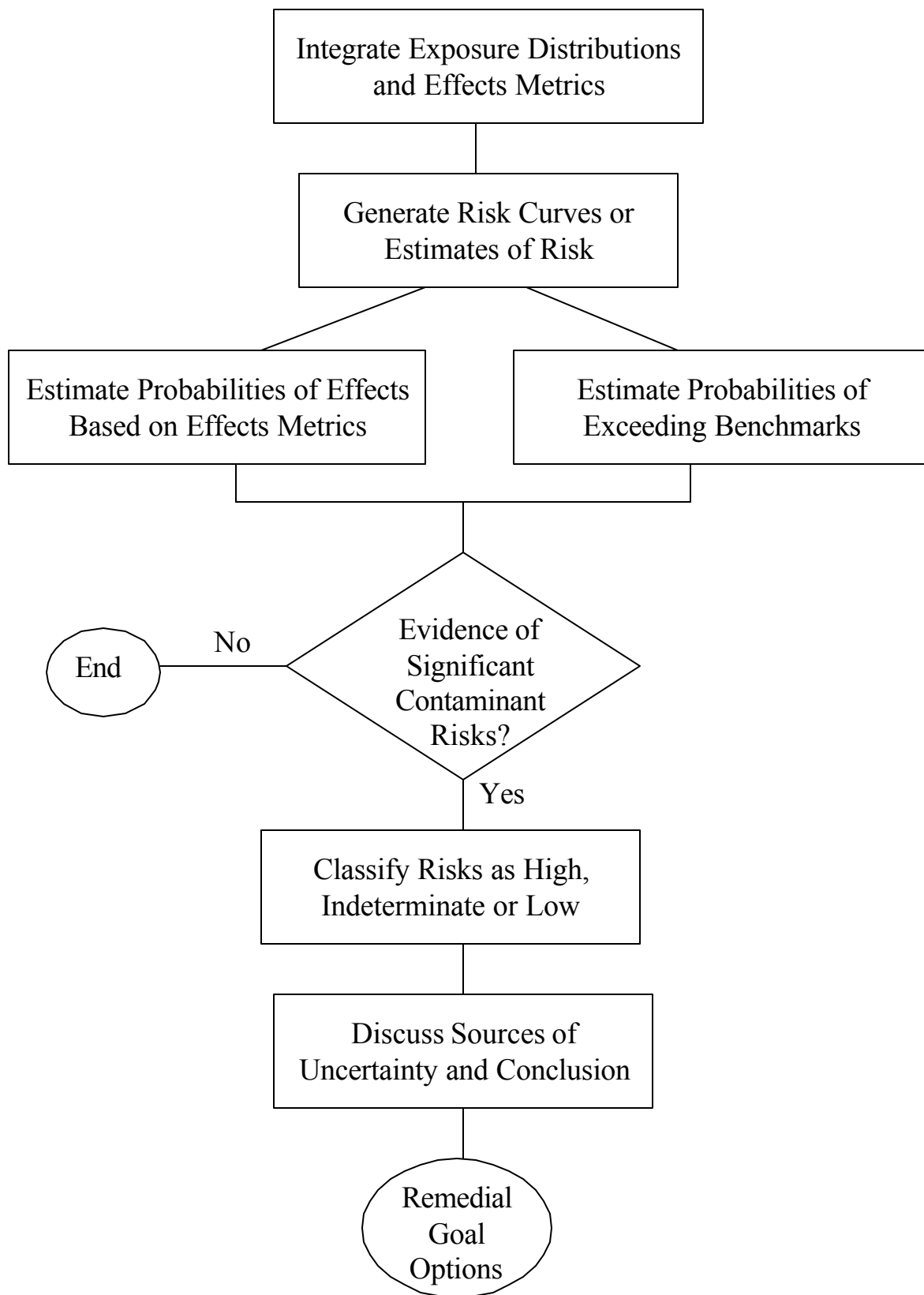
**Figure I2-1. Overview of approach used to assess exposure of mammals to contaminants of concern (COCs) in the Calcasieu Estuary.**



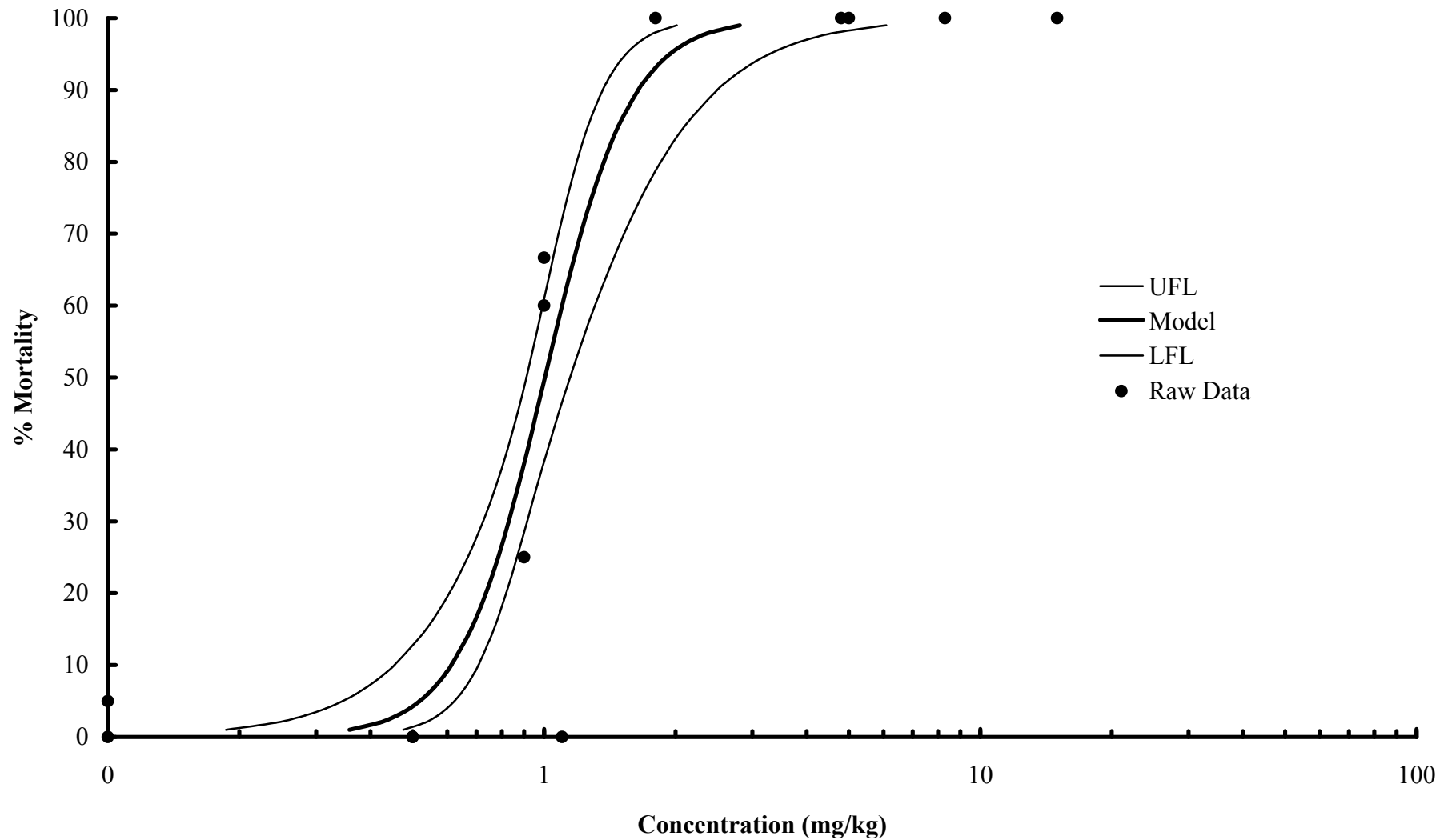
**Figure I2-2. Overview of approach used to assess the effects of mammals exposed to contaminants of concern (COCs) in the Calcasieu Estuary.**



**Figure I2-3. Overview of approach used to assess the risks of mammals exposed to contaminants of concern (COCs) in the Calcasieu Estuary.**



**Figure I2-4. Concentration-response curve with 95% fiducial limits for effects of dietary methylmercury on survival of female mink during chronic exposures. UFL and LFL are the upper and lower fiducial limits, respectively.**



**Figure 12-5. Dose-response curve with 95% fiducial limits for effects of dietary methylmercury on survival of female mink during chronic exposures. UFL and LFL are the upper and lower fiducial limits, respectively.**

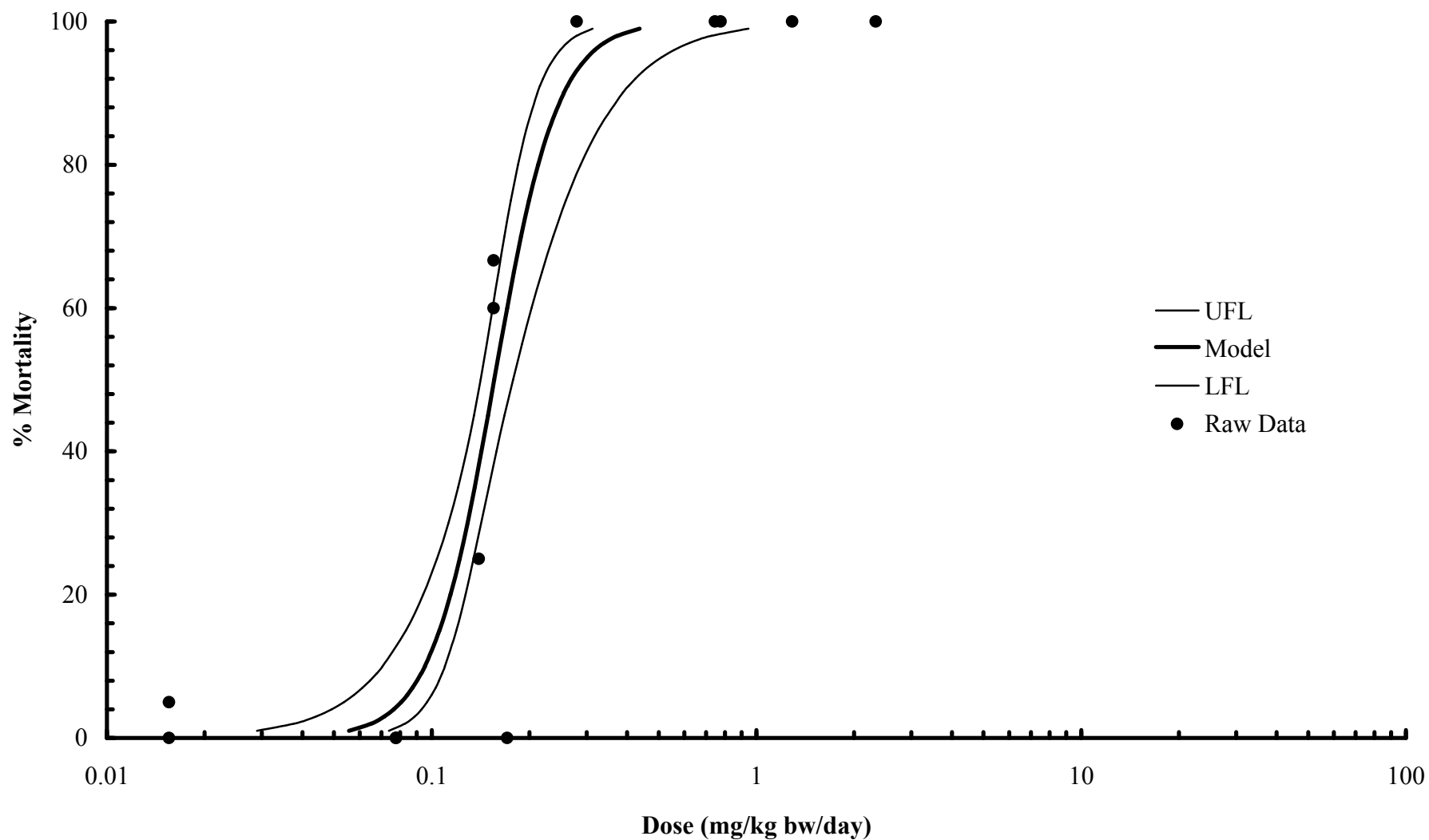


Figure I2-6. Dose-response curve for effects of dietary TCDD and equivalents on fecundity of female rat during chronic exposures.

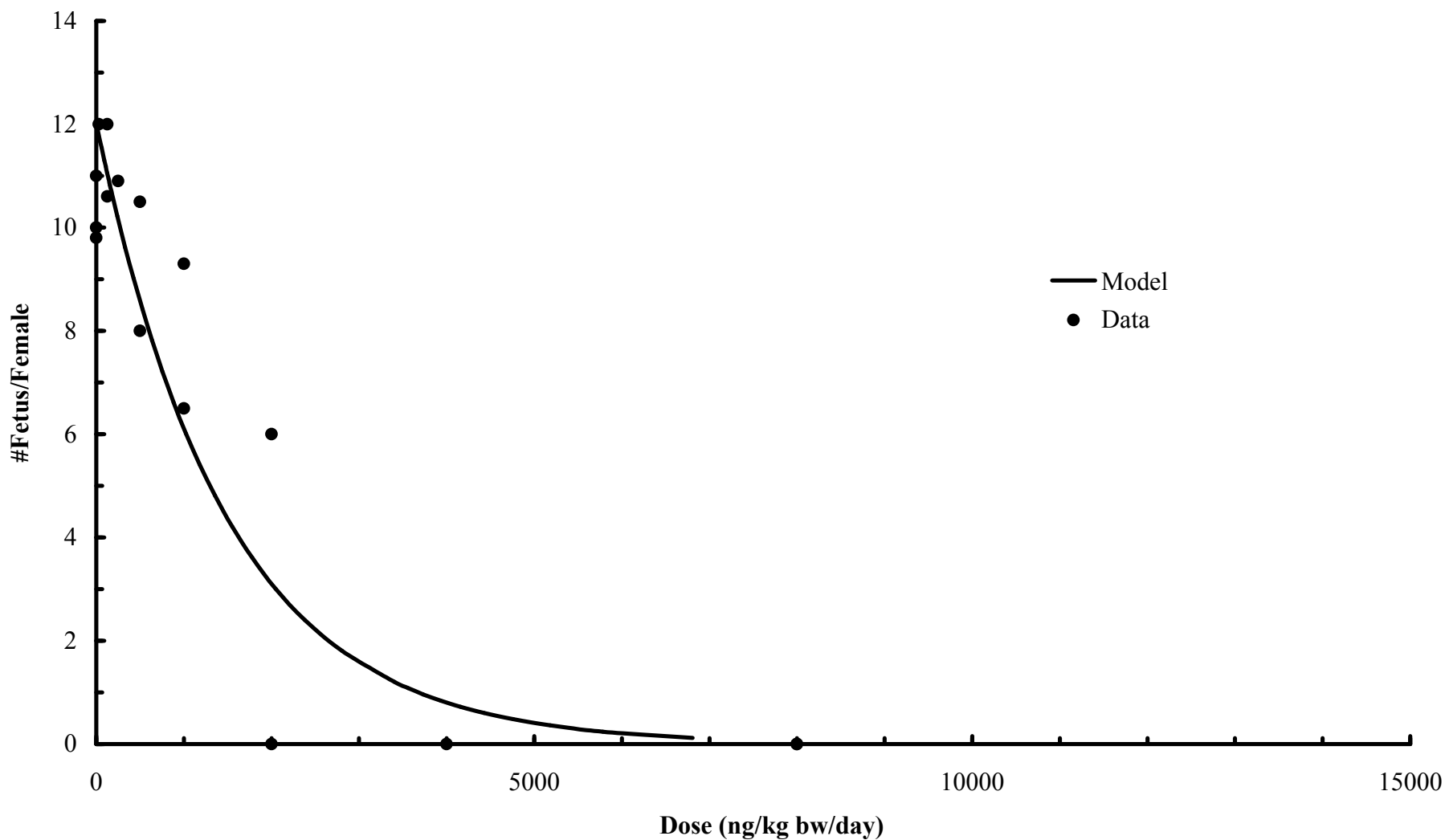
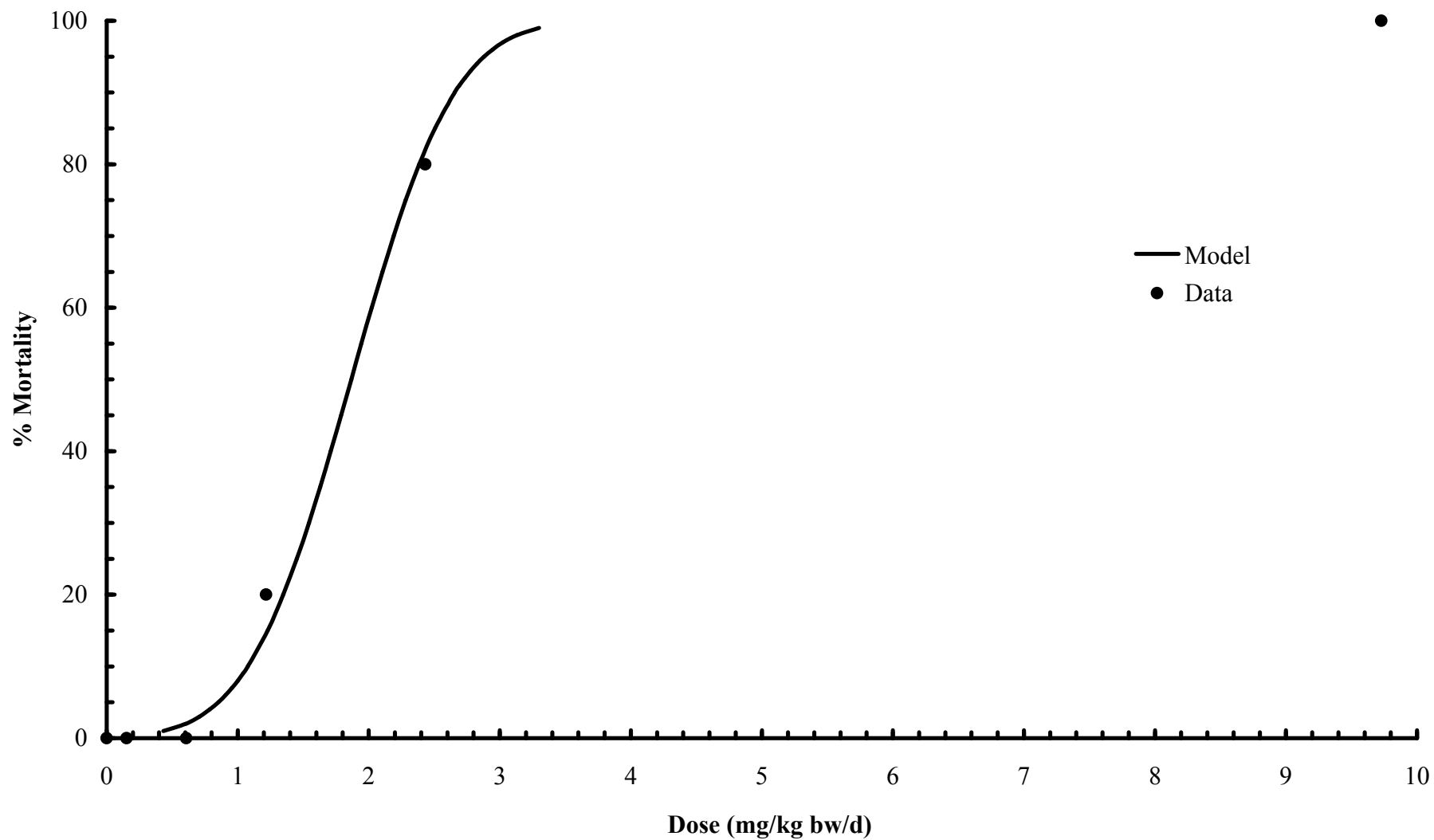
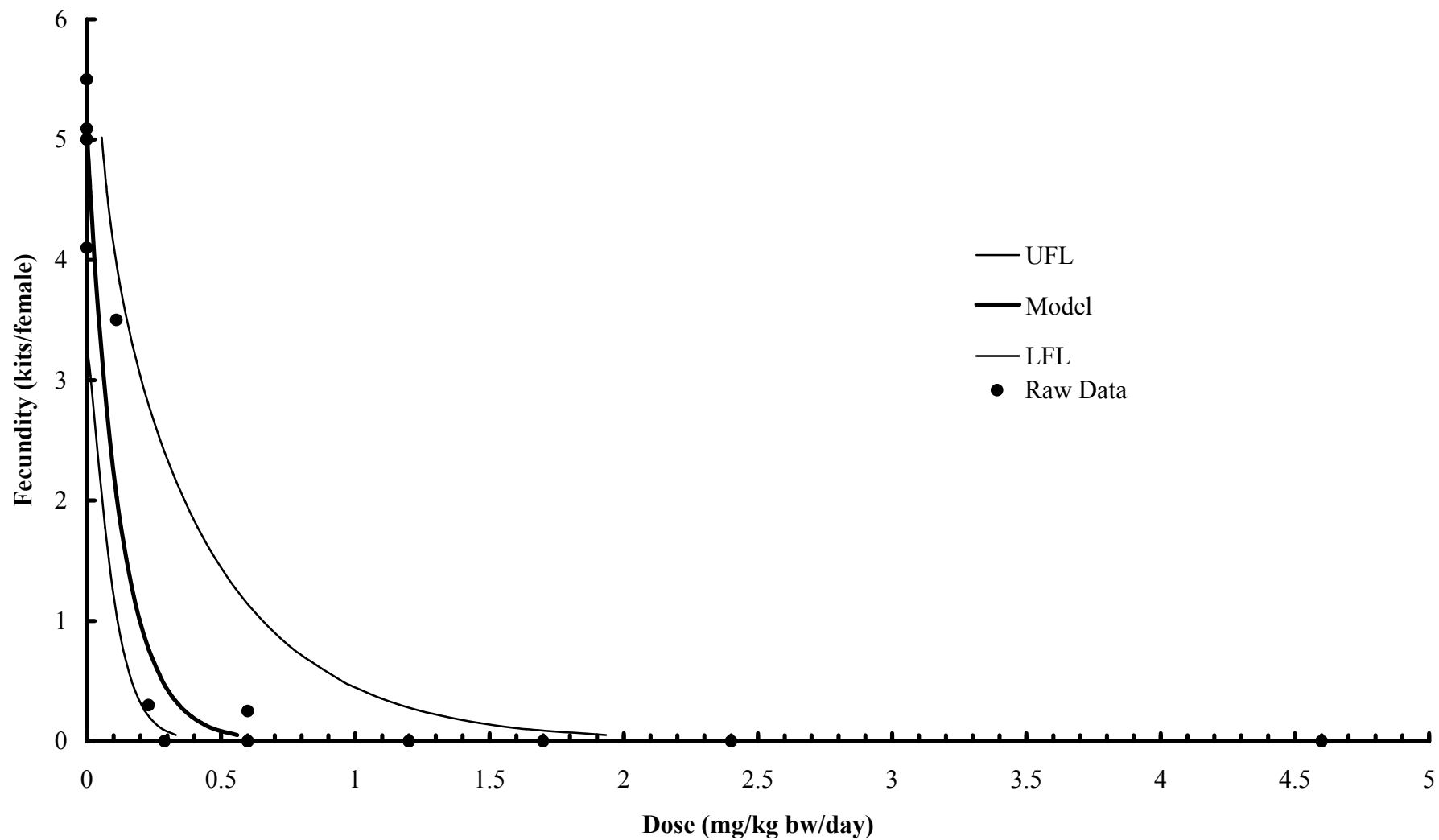


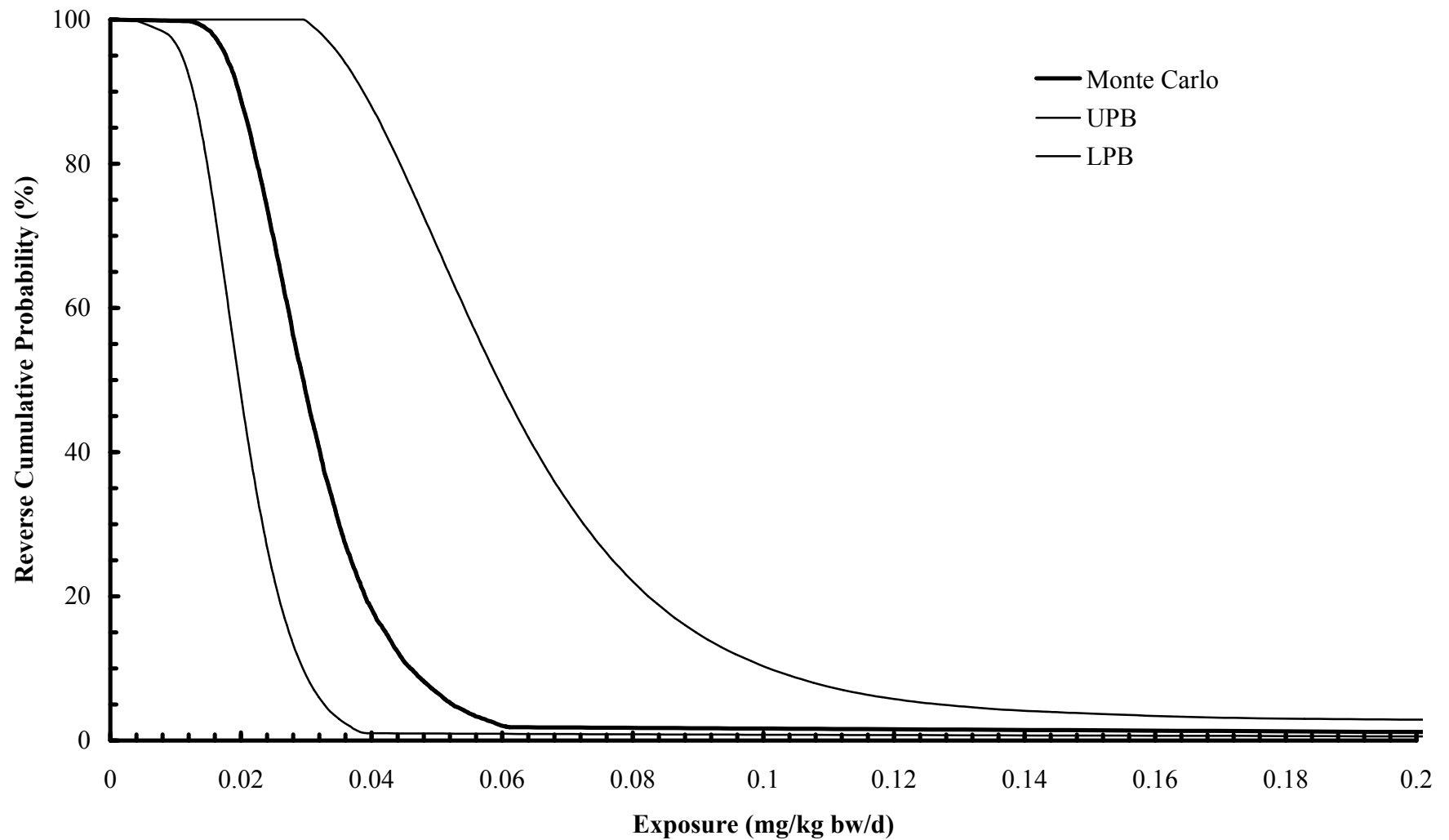
Figure I2-7. Dose-response curve for effects of dietary selenium on survival of rats during chronic exposures.



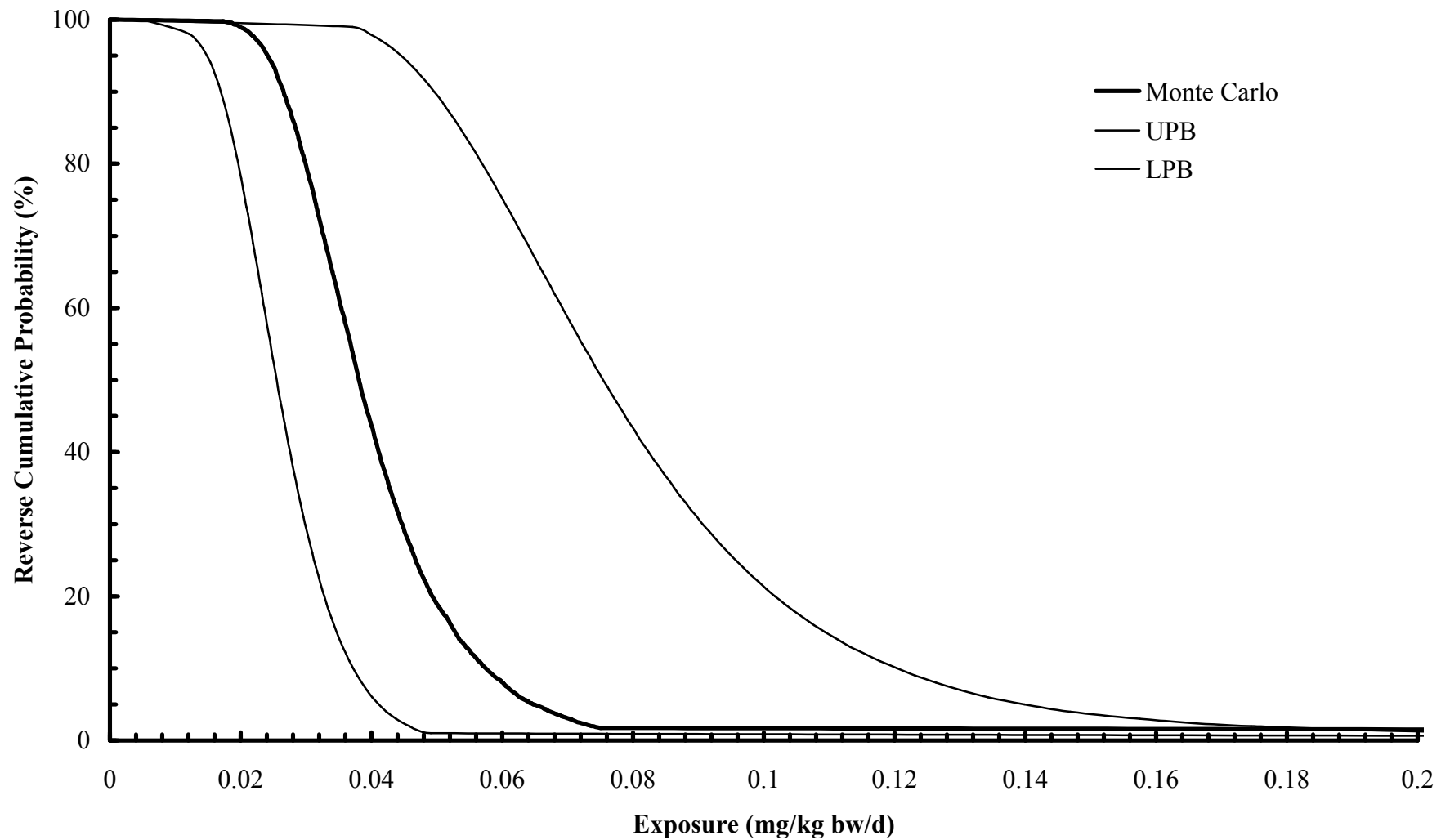
**Figure 12-8. Dose-response curve with 95% fiducial limits for effects of dietary total PCBs on fecundity of female mink during chronic exposures. UFL and LFL are the upper and lower fiducial limits, respectively.**



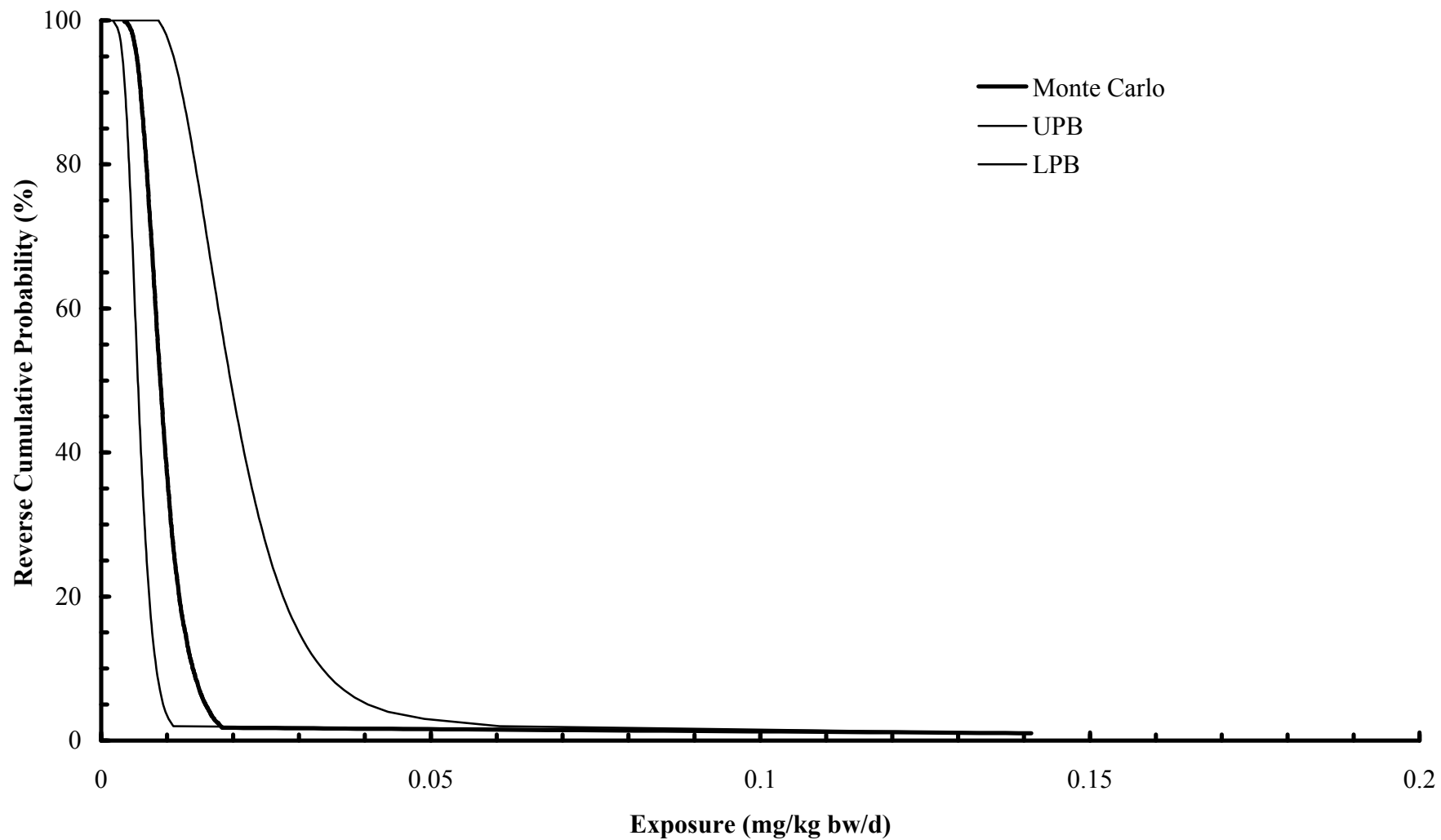
**Figure I2-9. Reverse cumulative probability distribution for average-sized piscivorous mammals exposed to mercury in Bayou d'Inde AOC (UPB and LPB are upper and lower probability bounds, respectively).**



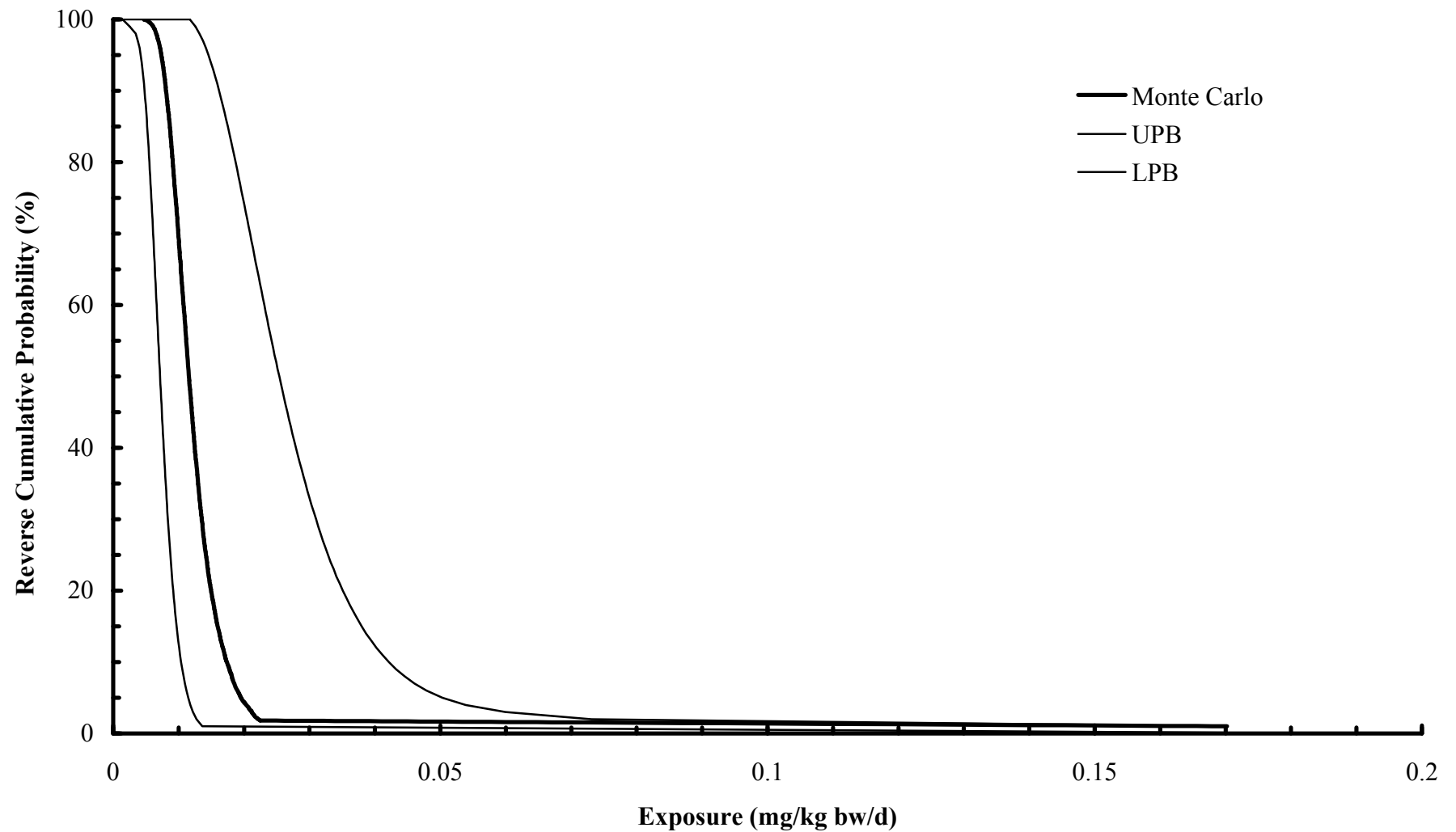
**Figure I2-10. Reverse cumulative probability distribution for small piscivorous mammals exposed to mercury in Bayou d'Inde AOC (UPB and LPB are upper and lower probability bounds, respectively).**



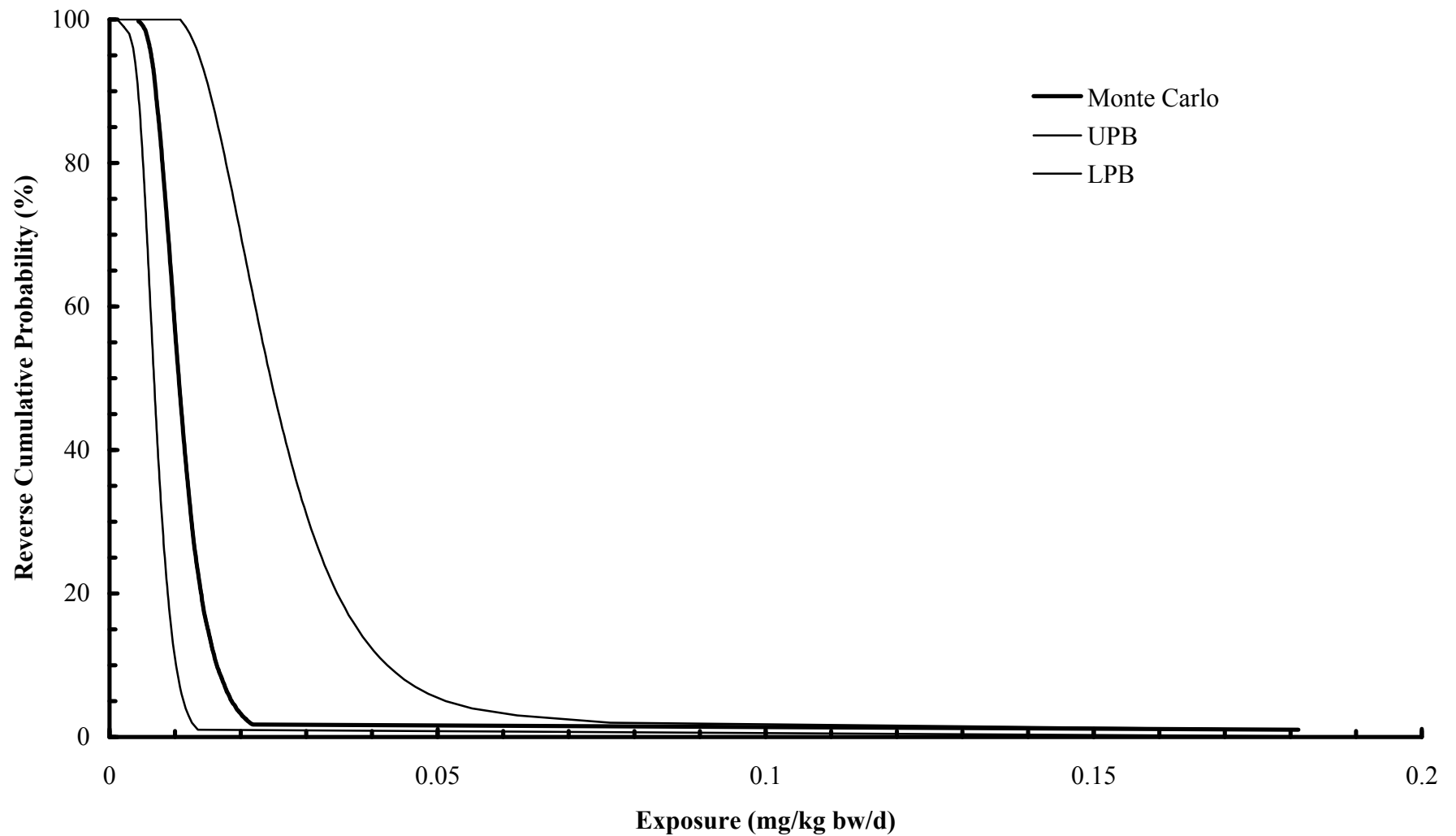
**Figure I2-11. Reverse cumulative probability distribution for average-sized piscivorous mammals exposed to mercury in Upper Calcasieu River AOC (UPB and LPB are upper and lower probability bounds, respectively).**



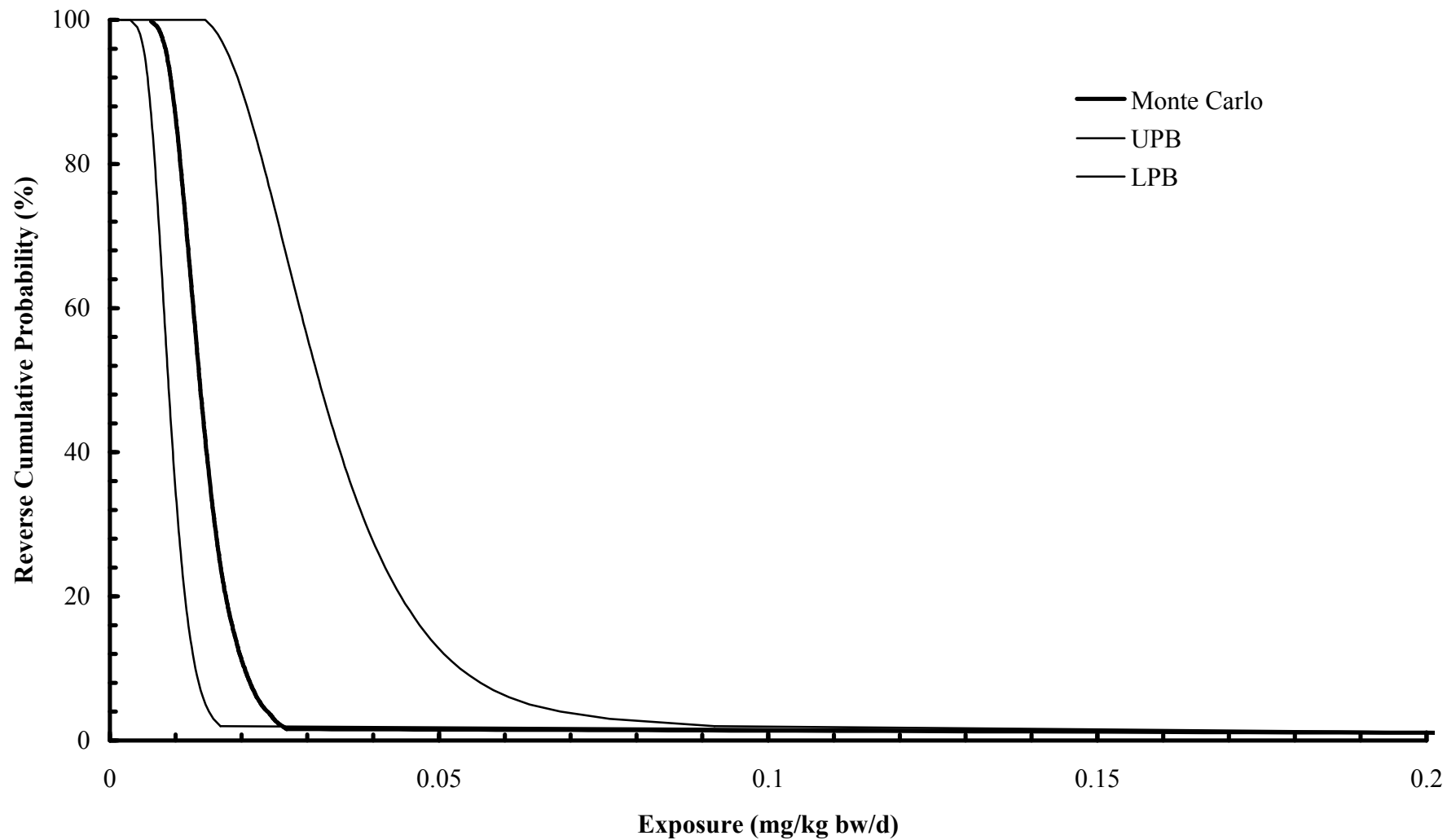
**Figure I2-12. Reverse cumulative probability distribution for small piscivorous mammals exposed to mercury in Upper Calcasieu River AOC (UPB and LPB are upper and lower probability bounds, respectively).**



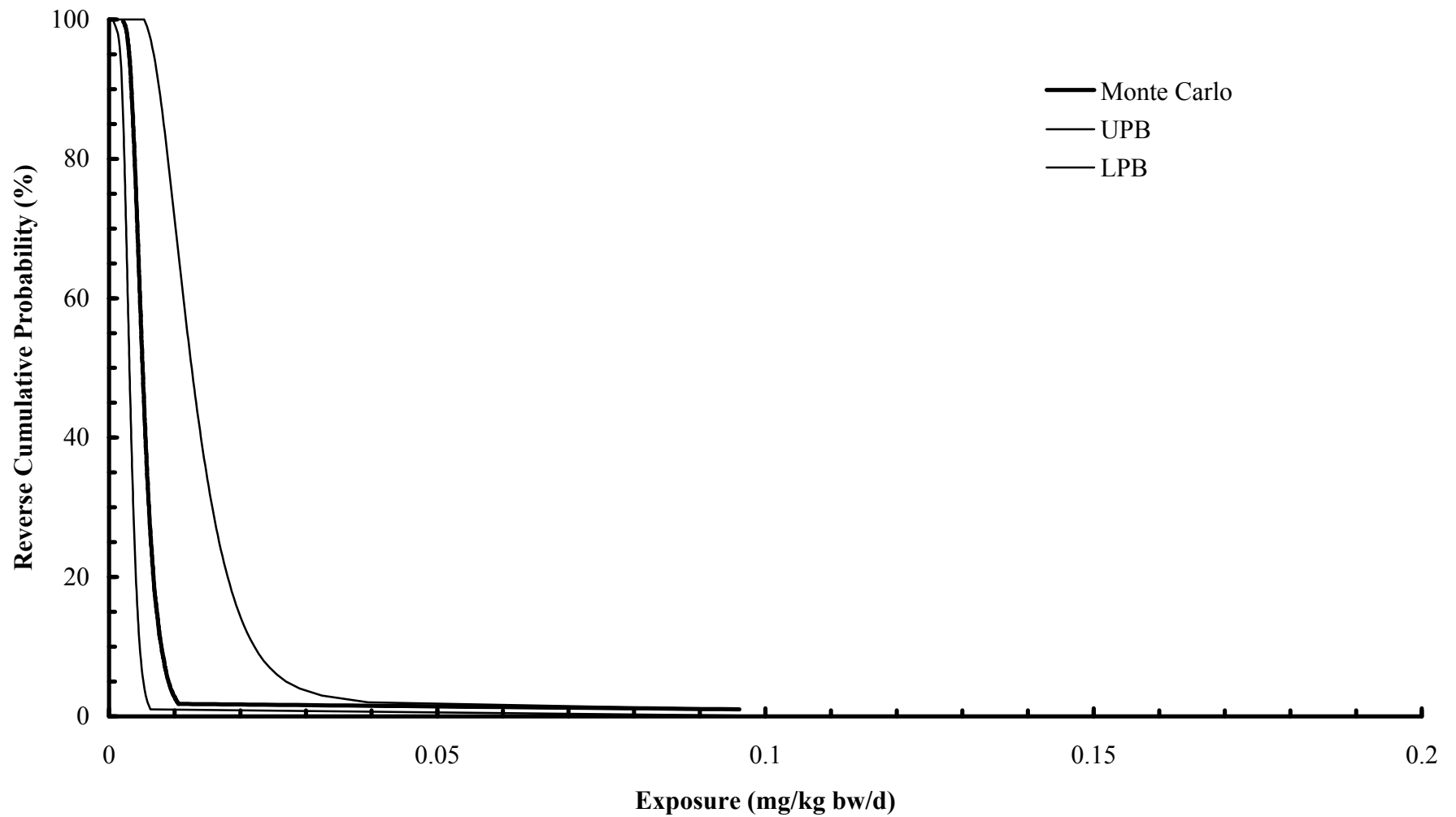
**Figure I2-13. Reverse cumulative probability distribution for average-sized piscivorous mammals exposed to mercury in Middle Calcasieu River AOC (UPB and LPB are upper and lower probability bounds, respectively).**



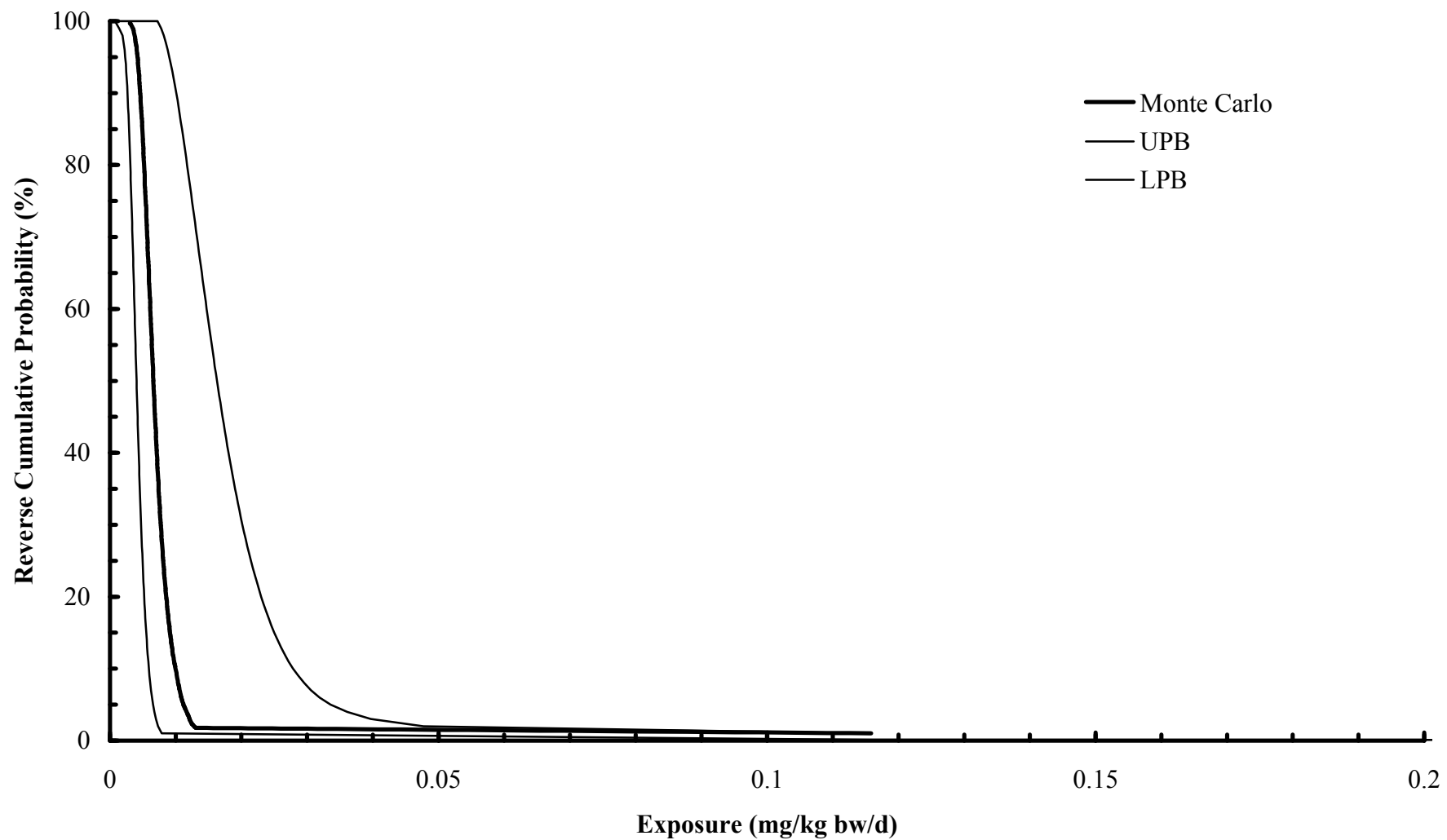
**Figure I2-14. Reverse cumulative probability distribution for small piscivorous mammals exposed to mercury in Middle Calcasieu River AOC (UPB and LPB are upper and lower probability bounds, respectively).**



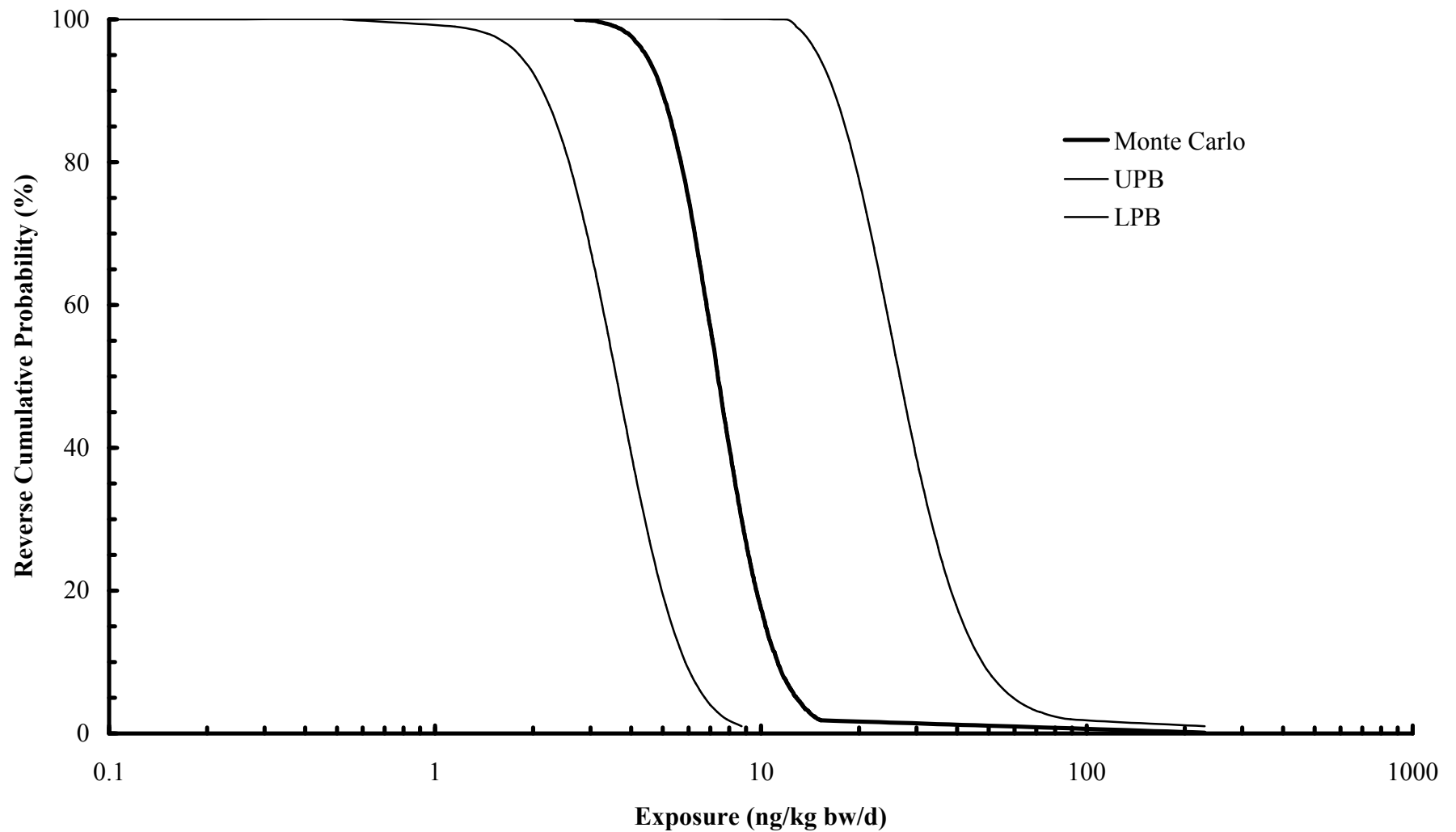
**Figure I2-15. Reverse cumulative probability distribution for average-sized piscivorous mammals exposed to mercury in reference areas (UPB and LPB are upper and lower probability bounds, respectively).**



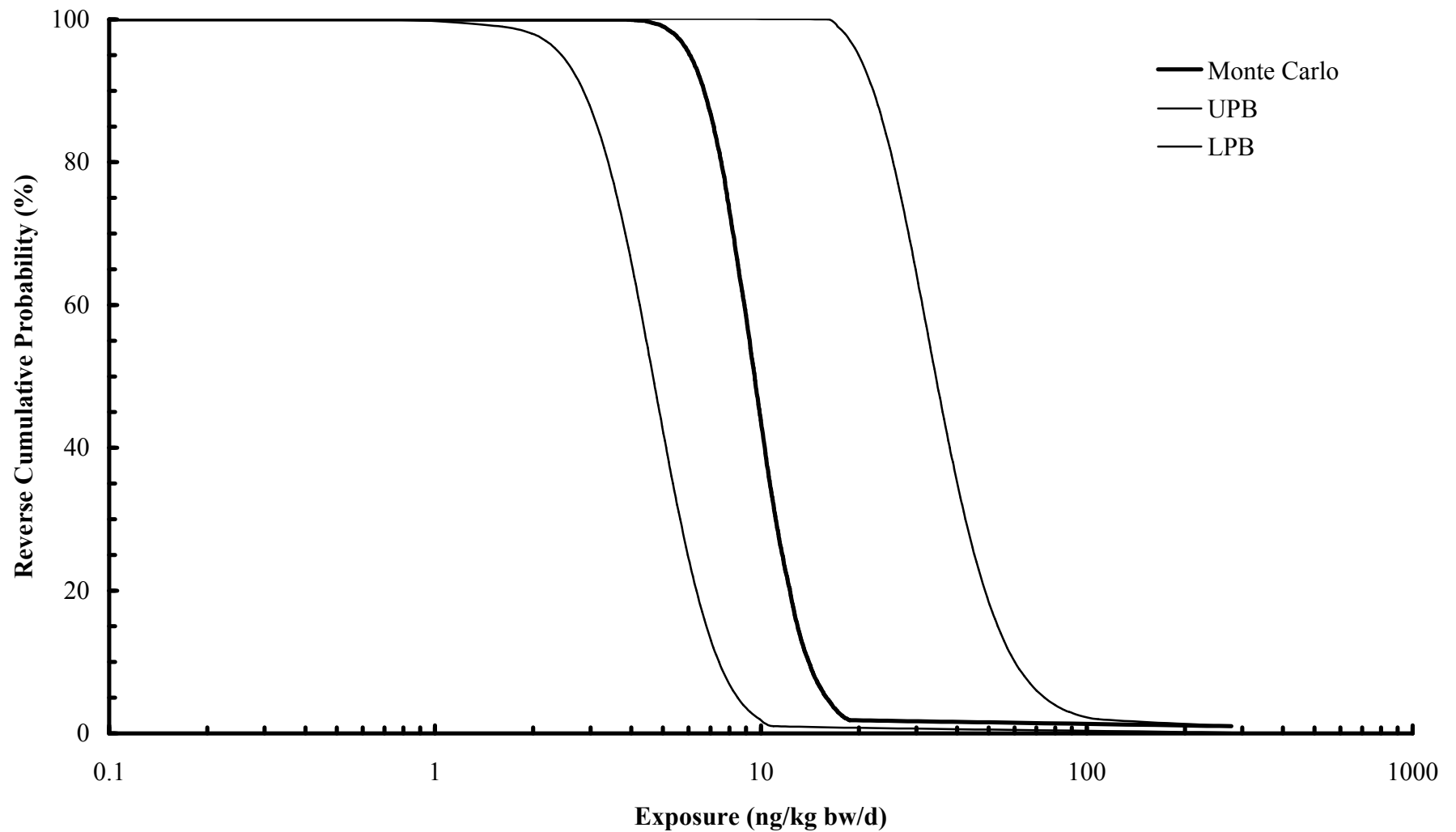
**Figure I2-16. Reverse cumulative probability distribution for small piscivorous mammals exposed to mercury in reference areas (UPB and LPB are upper and lower probability bounds, respectively).**



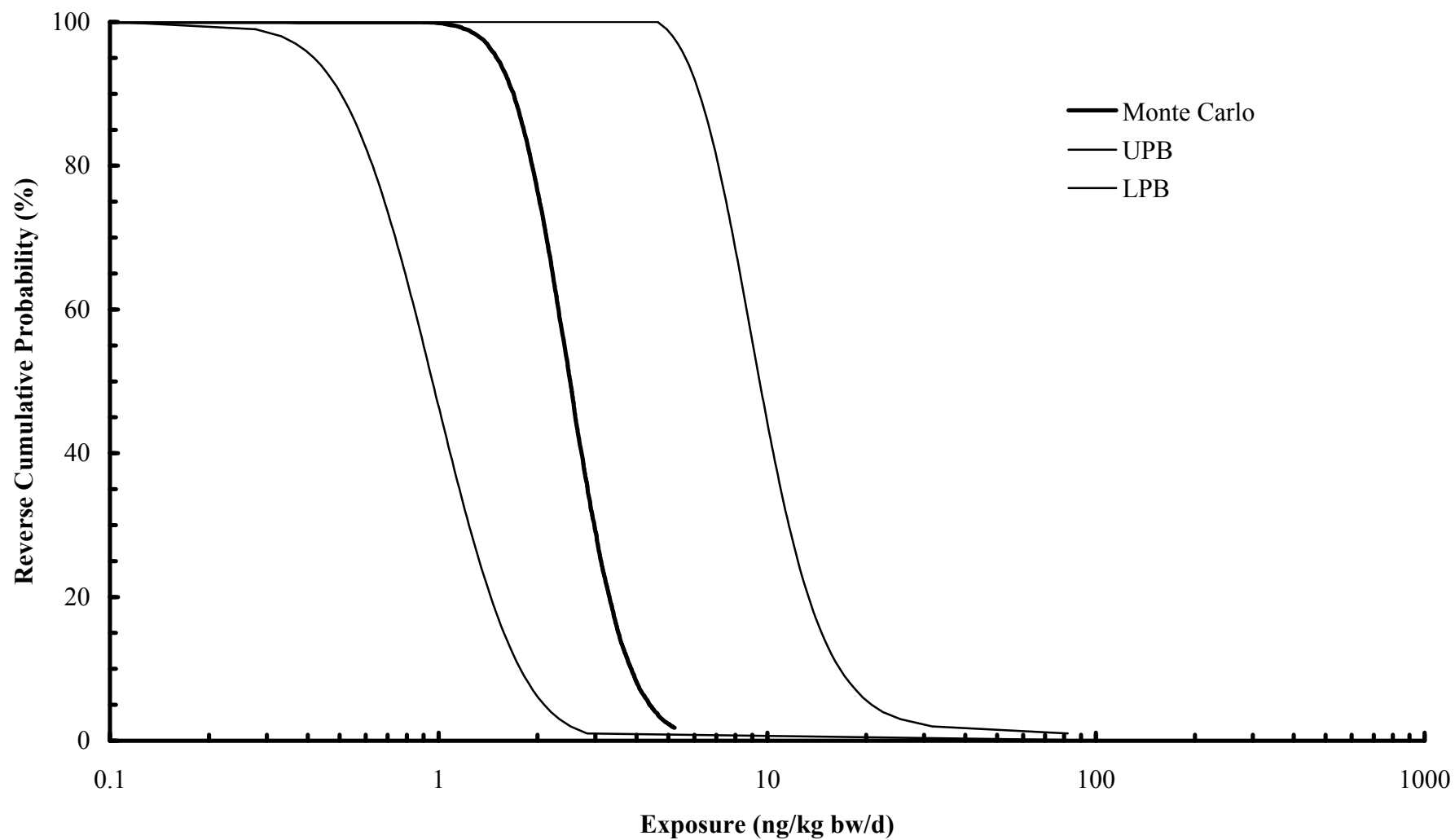
**Figure I2-17. Reverse cumulative probability distribution for average-sized piscivorous mammals exposed to TEQs in Bayou d'Inde AOC (UPB and LPB are upper and lower probability bounds, respectively).**



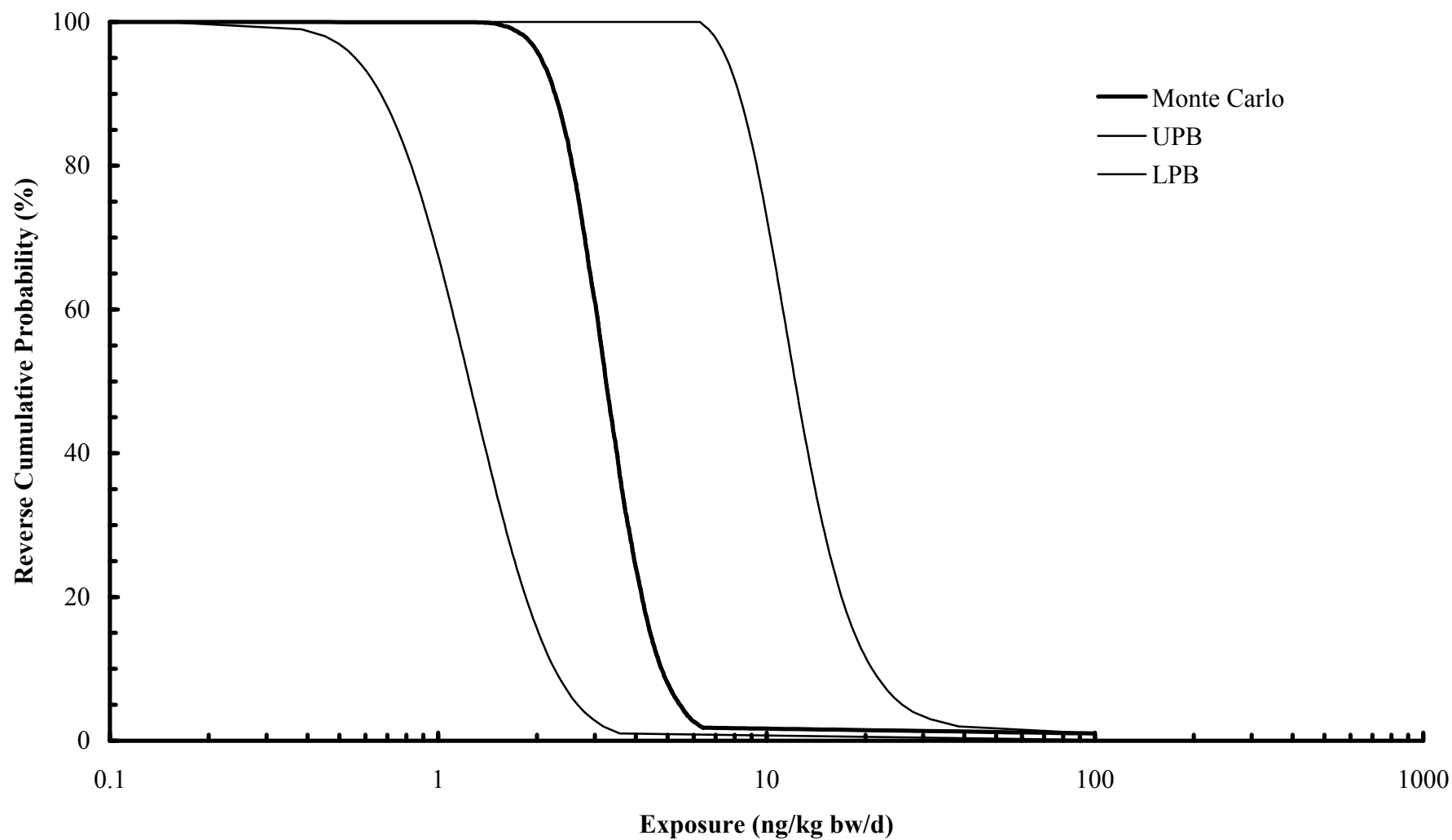
**Figure I2-18. Reverse cumulative probability distribution for small piscivorous mammals exposed to TEQs in Bayou d'Inde AOC (UPB and LPB are upper and lower probability bounds, respectively).**



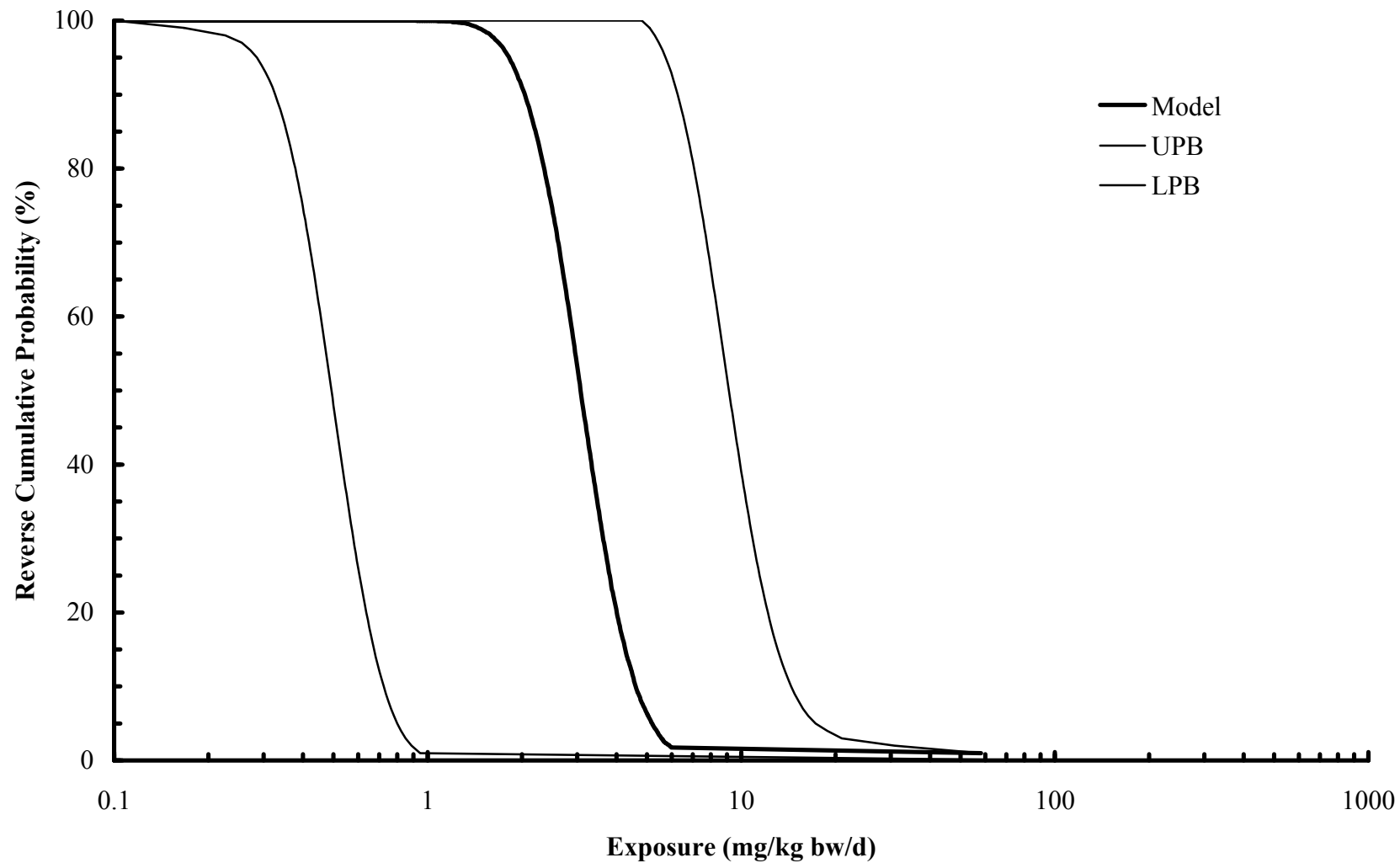
**Figure I2-19. Reverse cumulative probability distribution for average-sized piscivorous mammals exposed to TEQs in Upper Calcasieu River AOC (UPB and LPB are upper and lower probability bounds, respectively).**



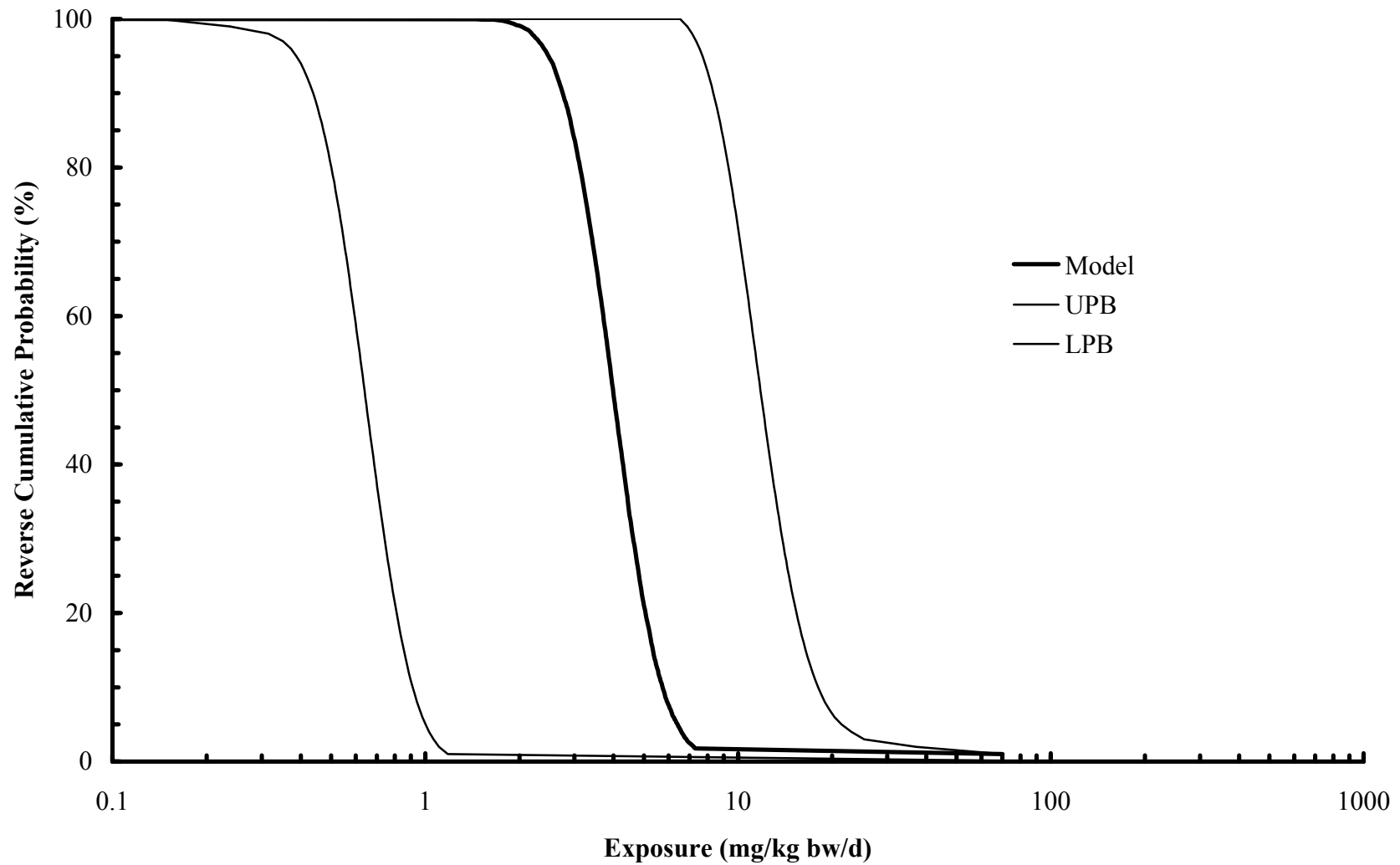
**Figure I2-20. Reverse cumulative probability distribution for small piscivorous mammals exposed to TEQs in Upper Calcasieu River AOC (UPB and LPB are upper and lower probability bounds, respectively).**



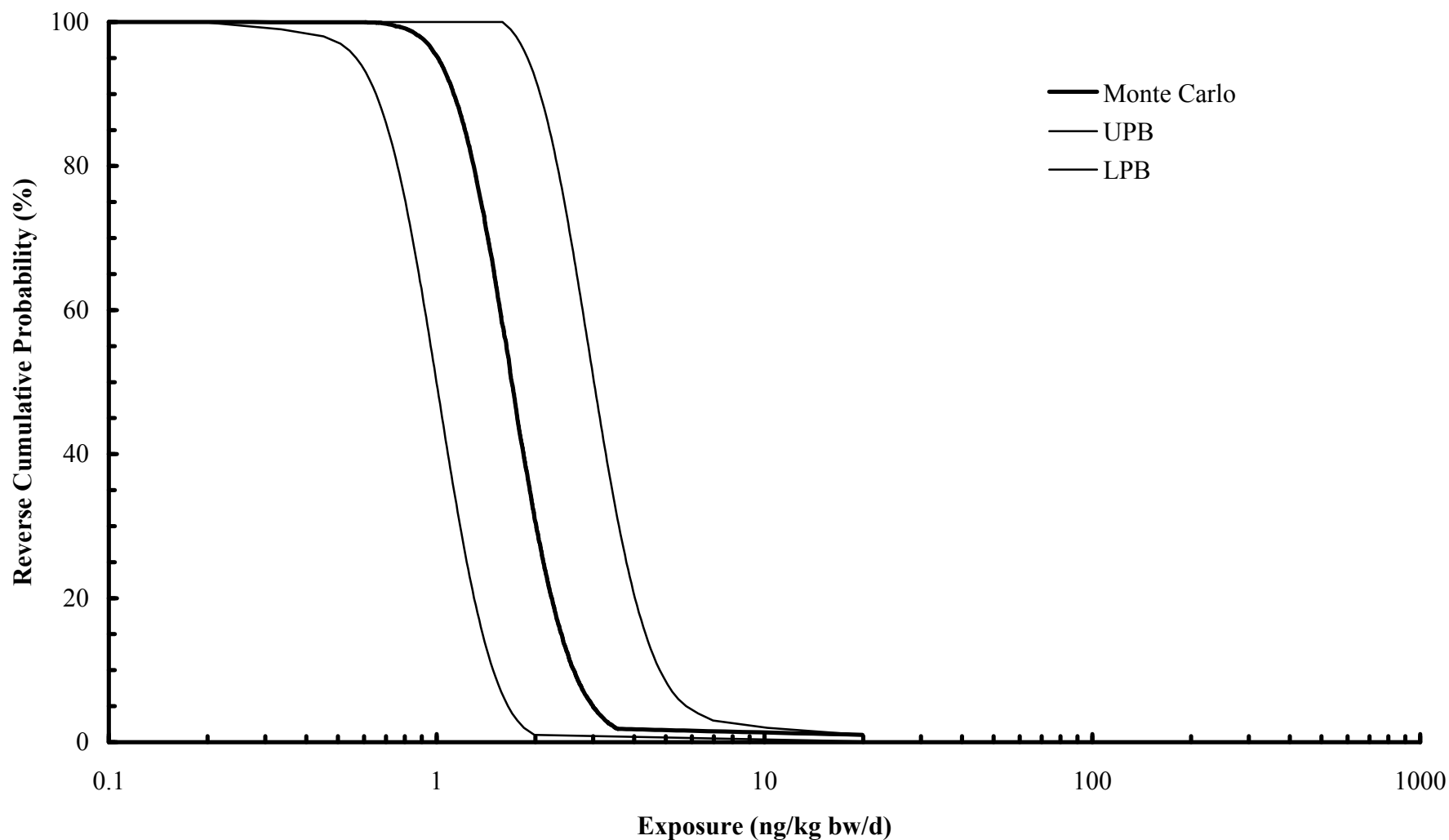
**Figure I2-21. Reverse cumulative probability distribution for average-sized piscivorous mammals exposed to TEQs in Middle Calcasieu River AOC (UPB and LPB are upper and lower probability bounds, respectively).**



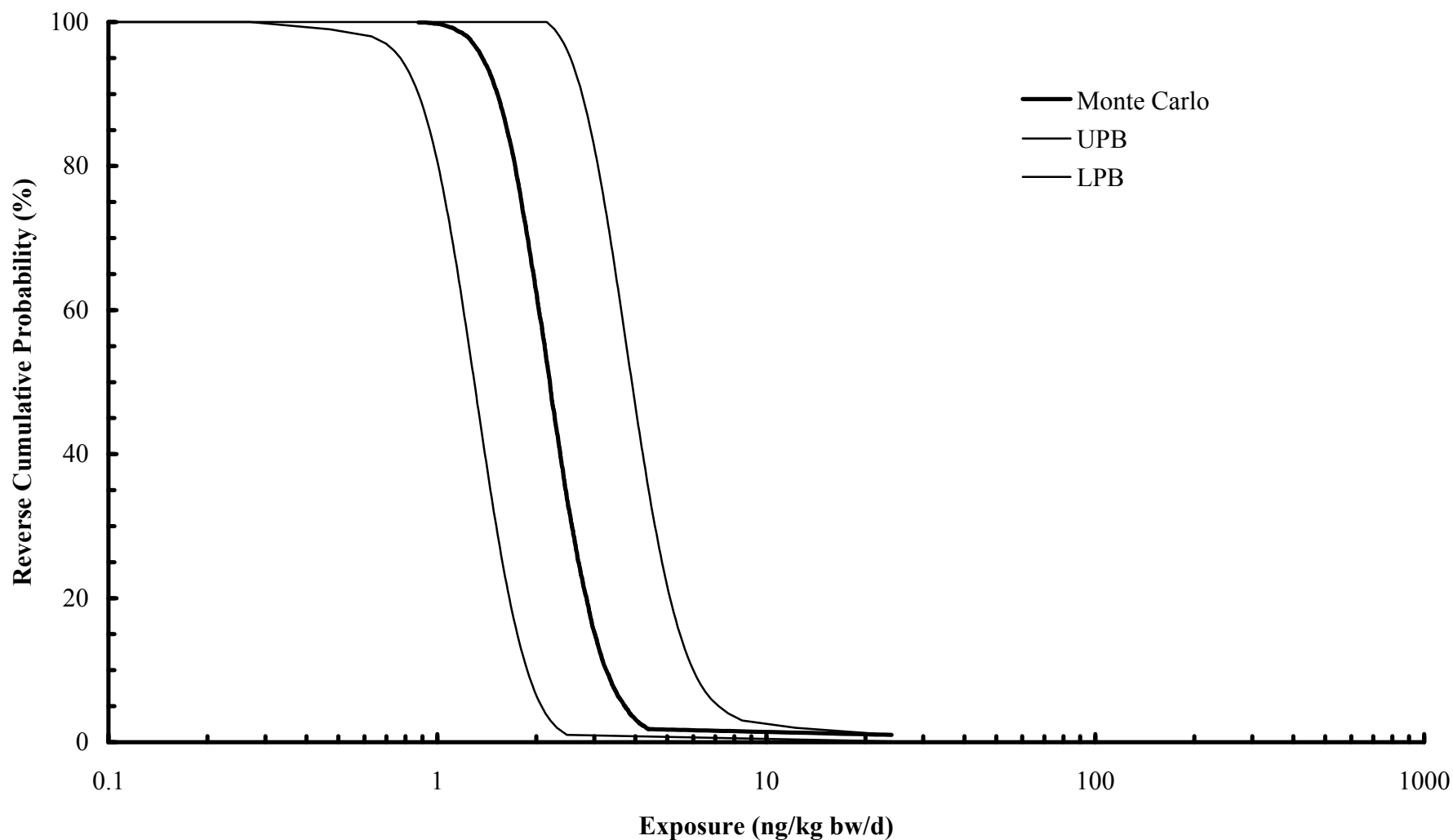
**Figure I2-22. Reverse cumulative probability distribution for small piscivorous mammals exposed to TEQs in Middle Calcasieu River AOC (UPB and LPB are upper and lower probability bounds, respectively).**



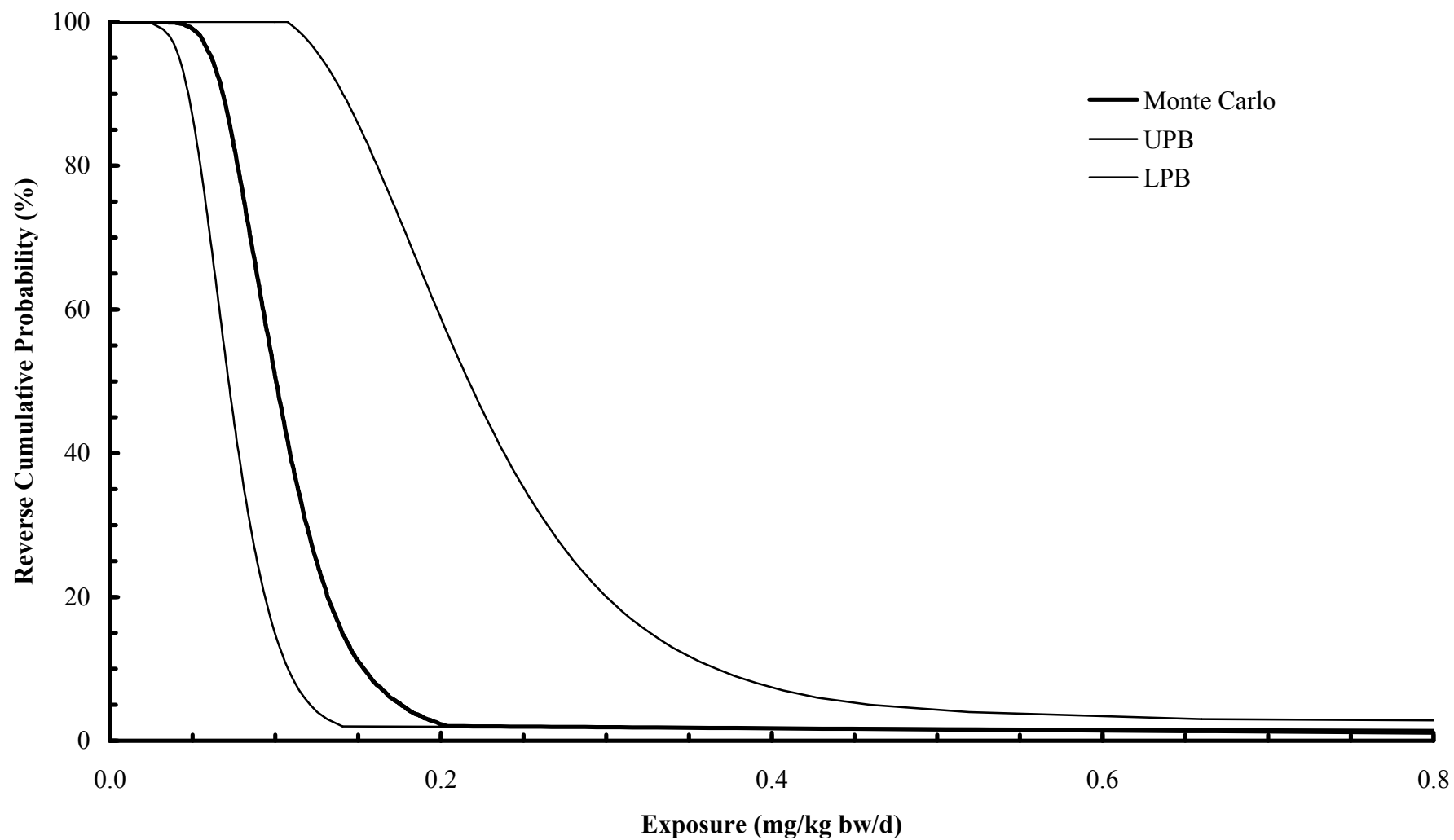
**Figure I2-23. Reverse cumulative probability distribution for average-sized piscivorous mammals exposed to TEQs in reference areas (UPB and LPB are upper and lower probability bounds, respectively).**



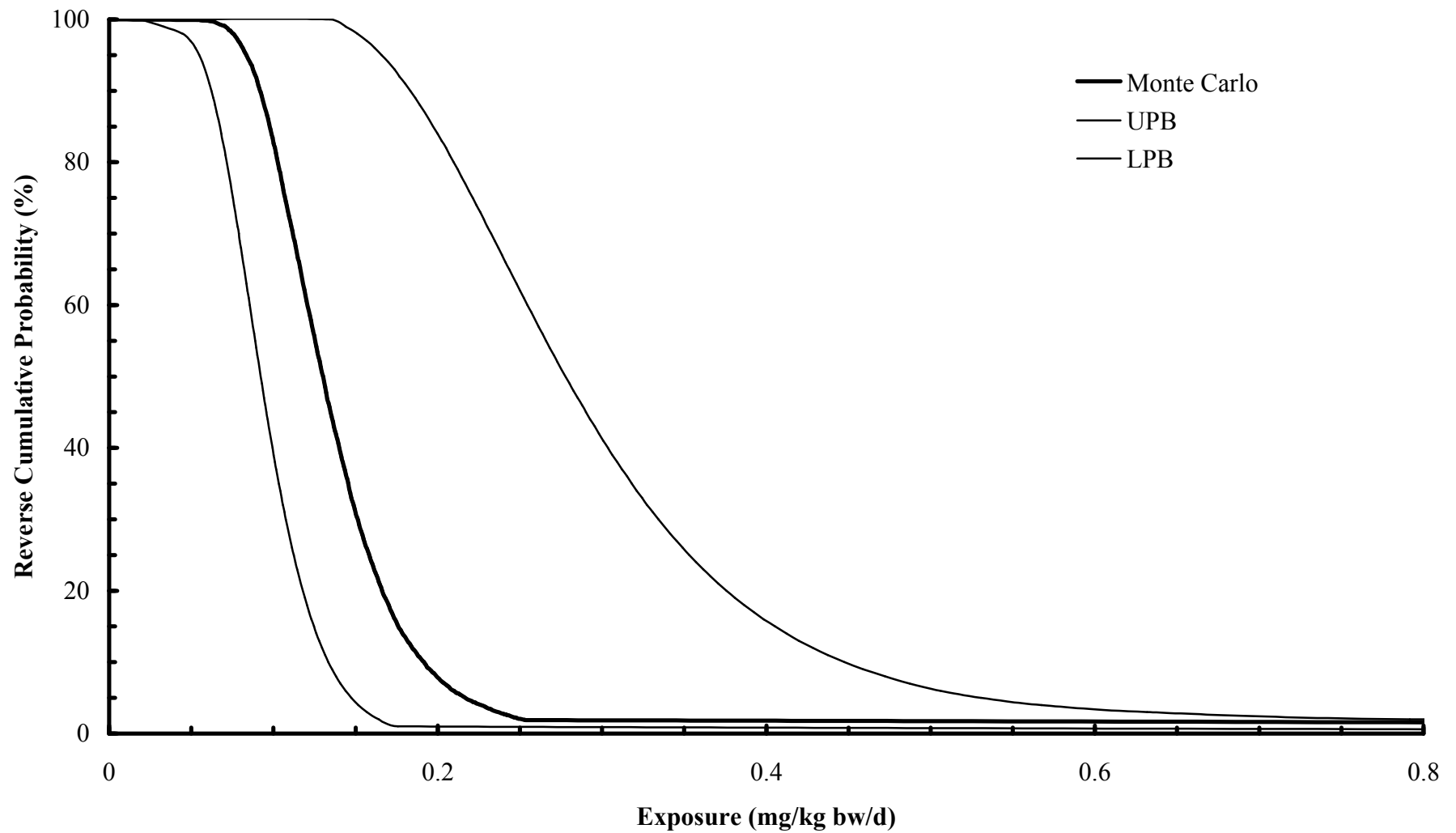
**Figure I2-24. Reverse cumulative probability distribution for small piscivorous mammals exposed to TEQs in reference areas (UPB and LPB are upper and lower probability bounds, respectively).**



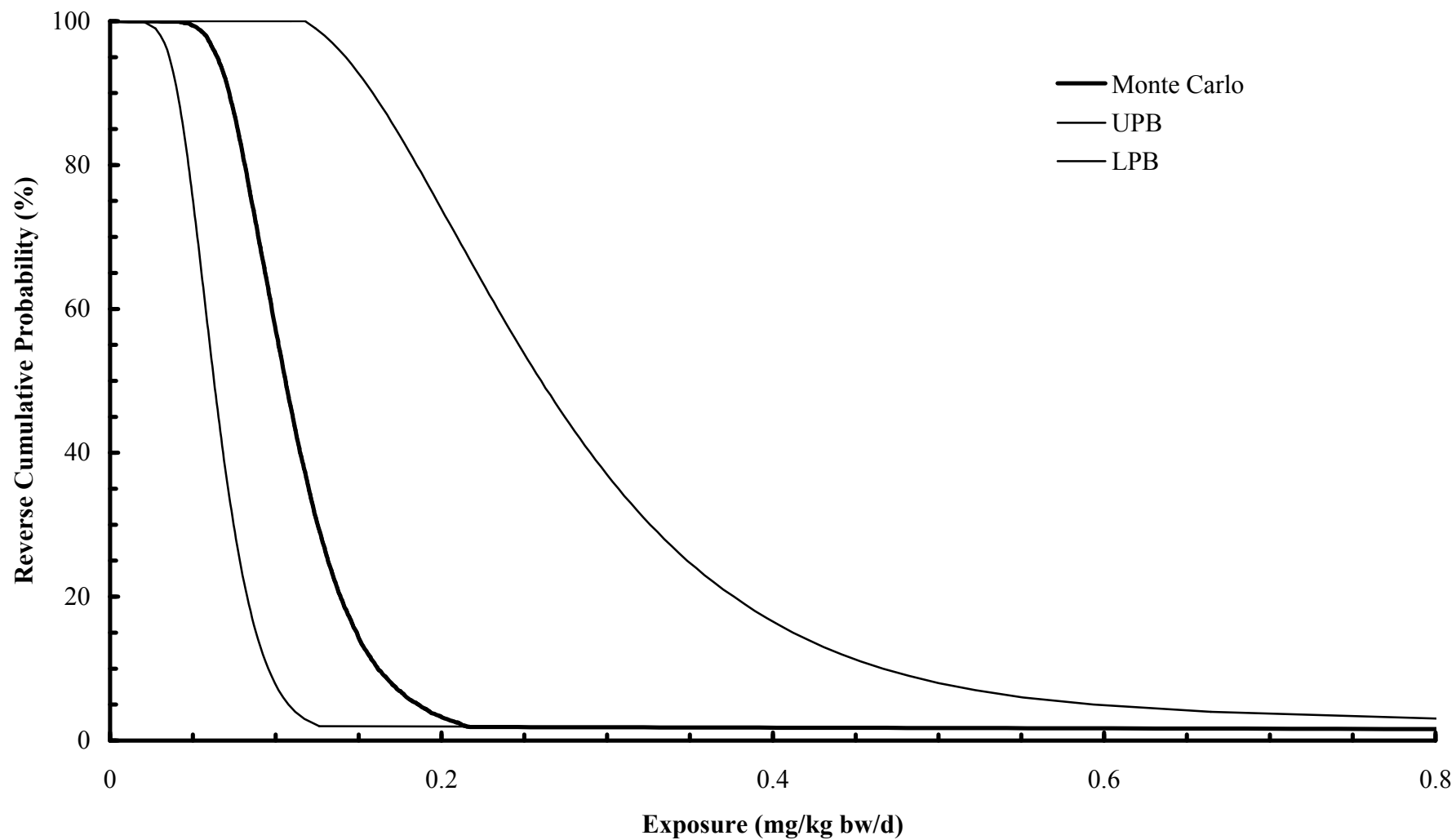
**Figure I2-25. Reverse cumulative probability distribution for average-sized piscivorous mammals exposed to selenium in Bayou d'Inde AOC (UPB and LPB are upper and lower probability bounds, respectively).**



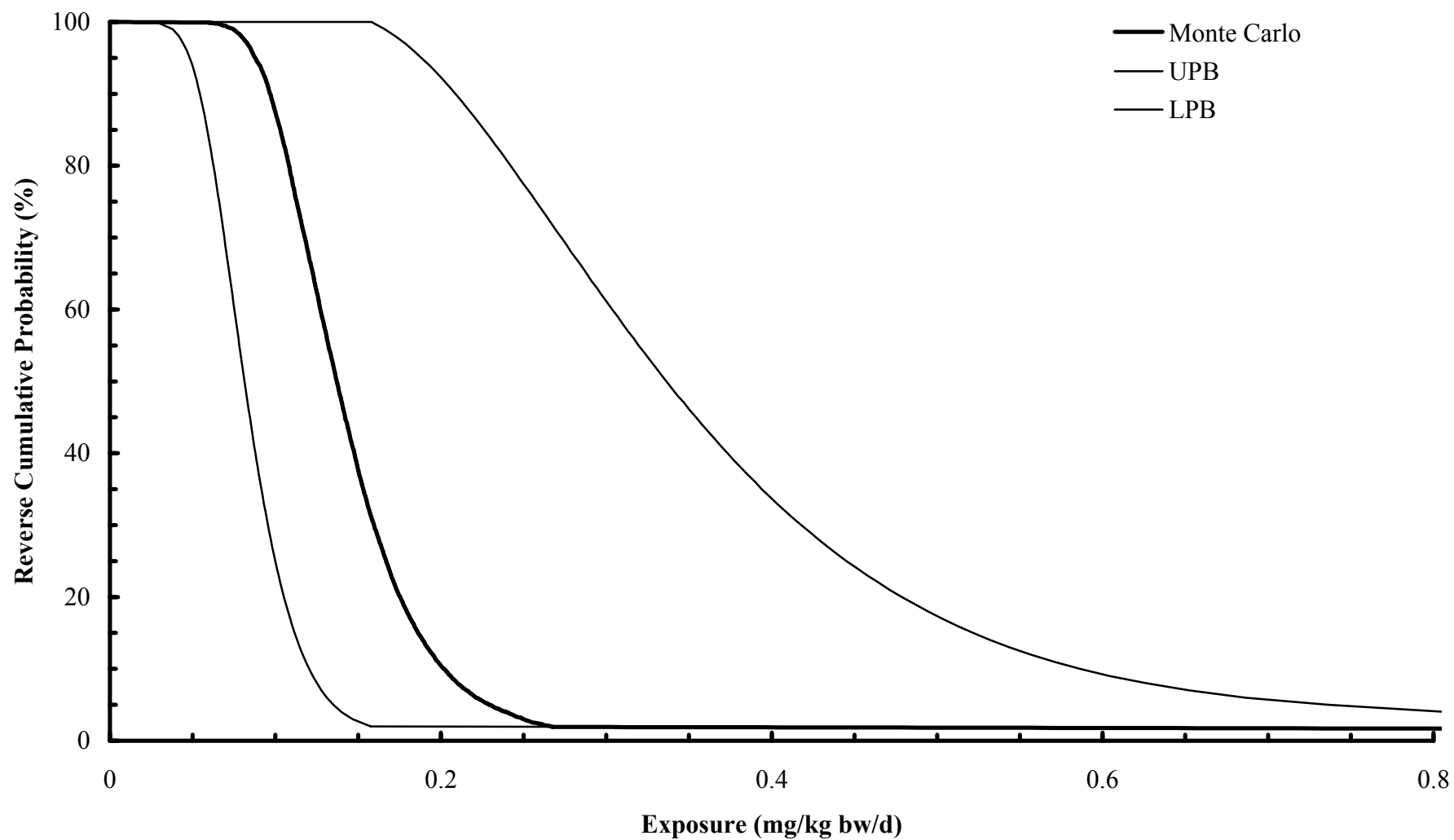
**Figure I2-26. Reverse cumulative probability distribution for small piscivorous mammals exposed to selenium in Bayou d'Inde AOC (UPB and LPB are upper and lower probability bounds, respectively).**



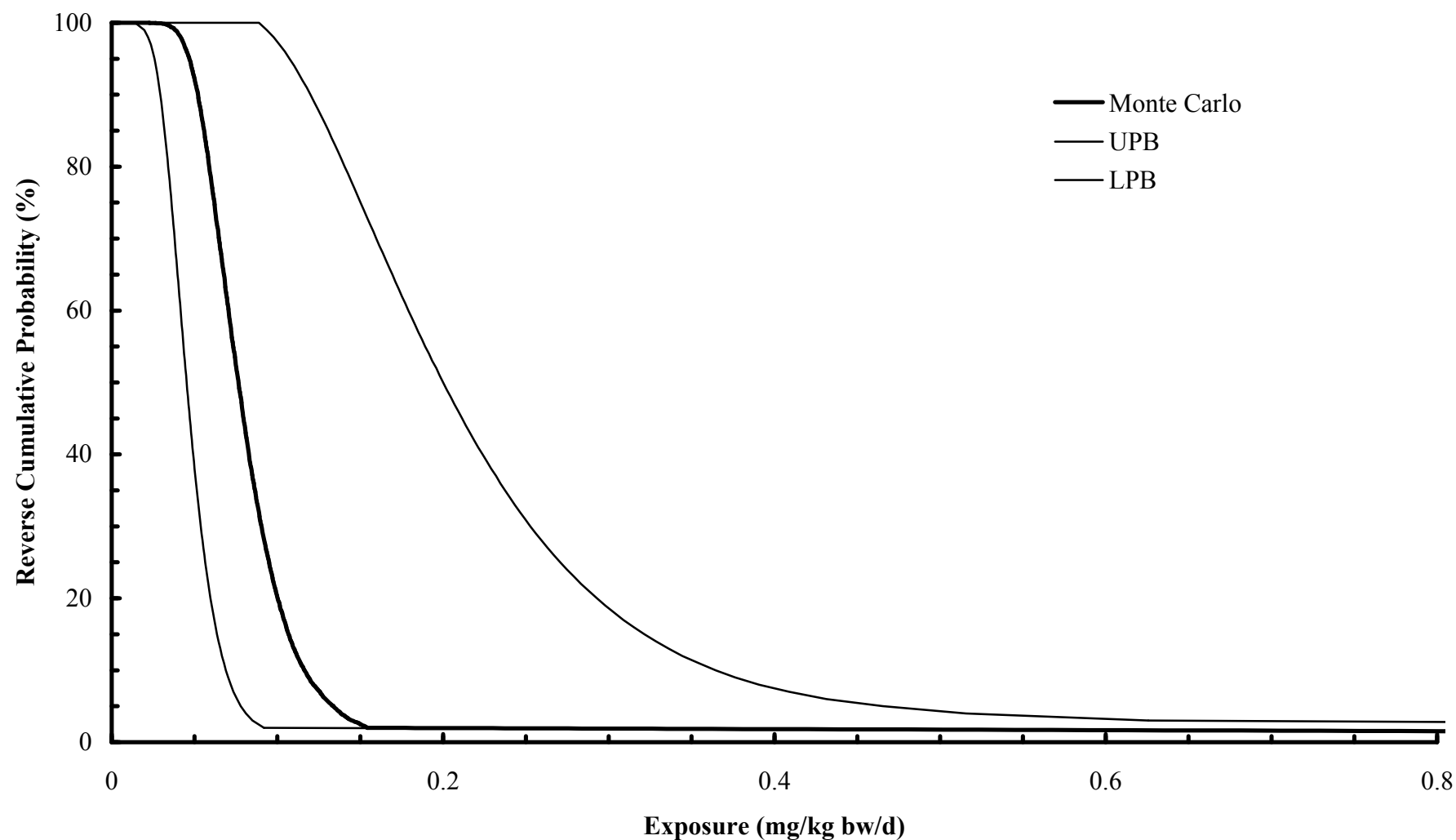
**Figure I2-27. Reverse cumulative probability distribution for average-sized piscivorous mammals exposed to selenium in Middle Calcasieu River AOC (UPB and LPB are upper and lower probability bounds, respectively).**



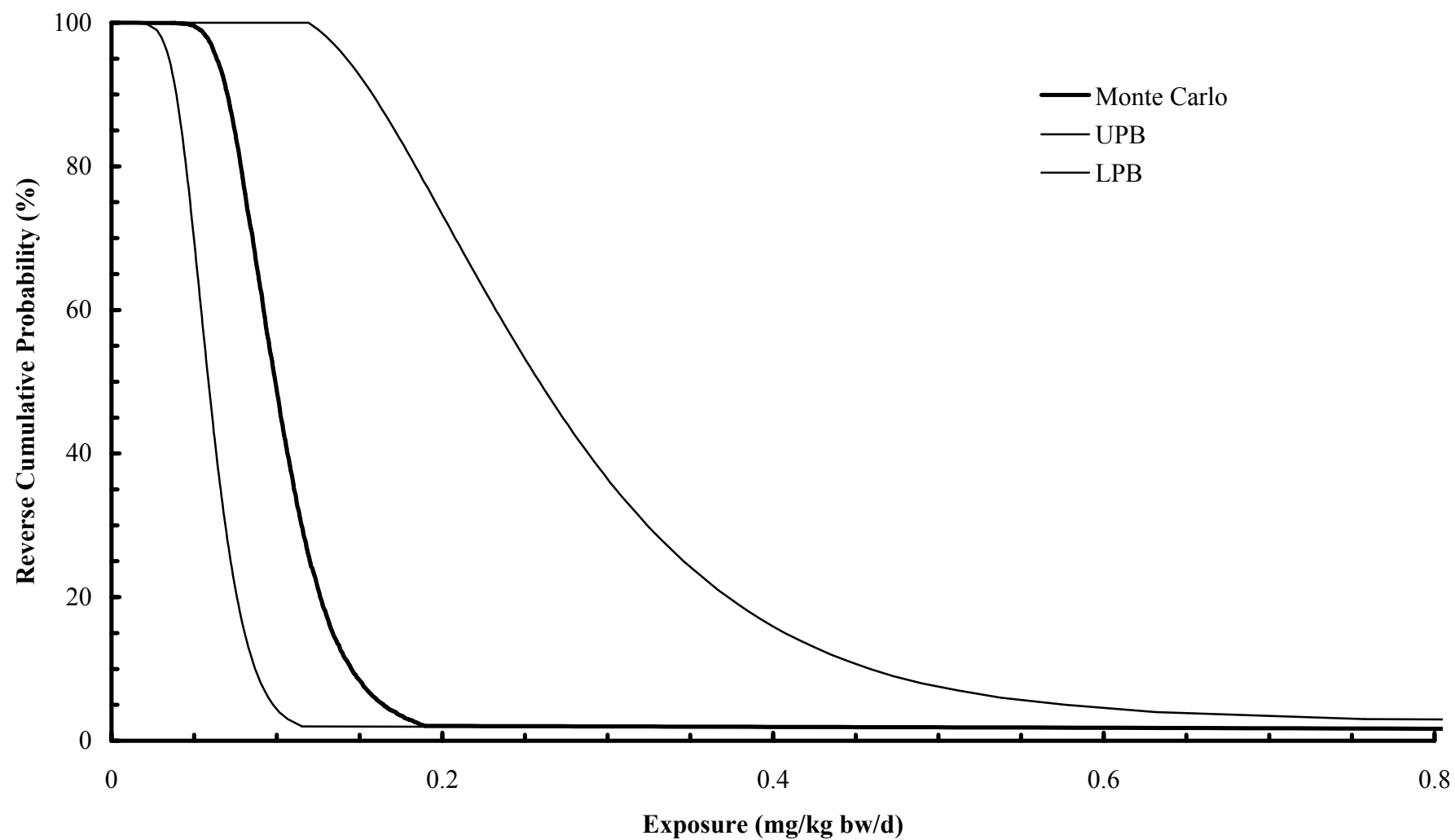
**Figure I2-28. Reverse cumulative probability distribution for small piscivorous mammals exposed to selenium in Middle Calcasieu River AOC (UPB and LPB are upper and lower probability bounds, respectively).**



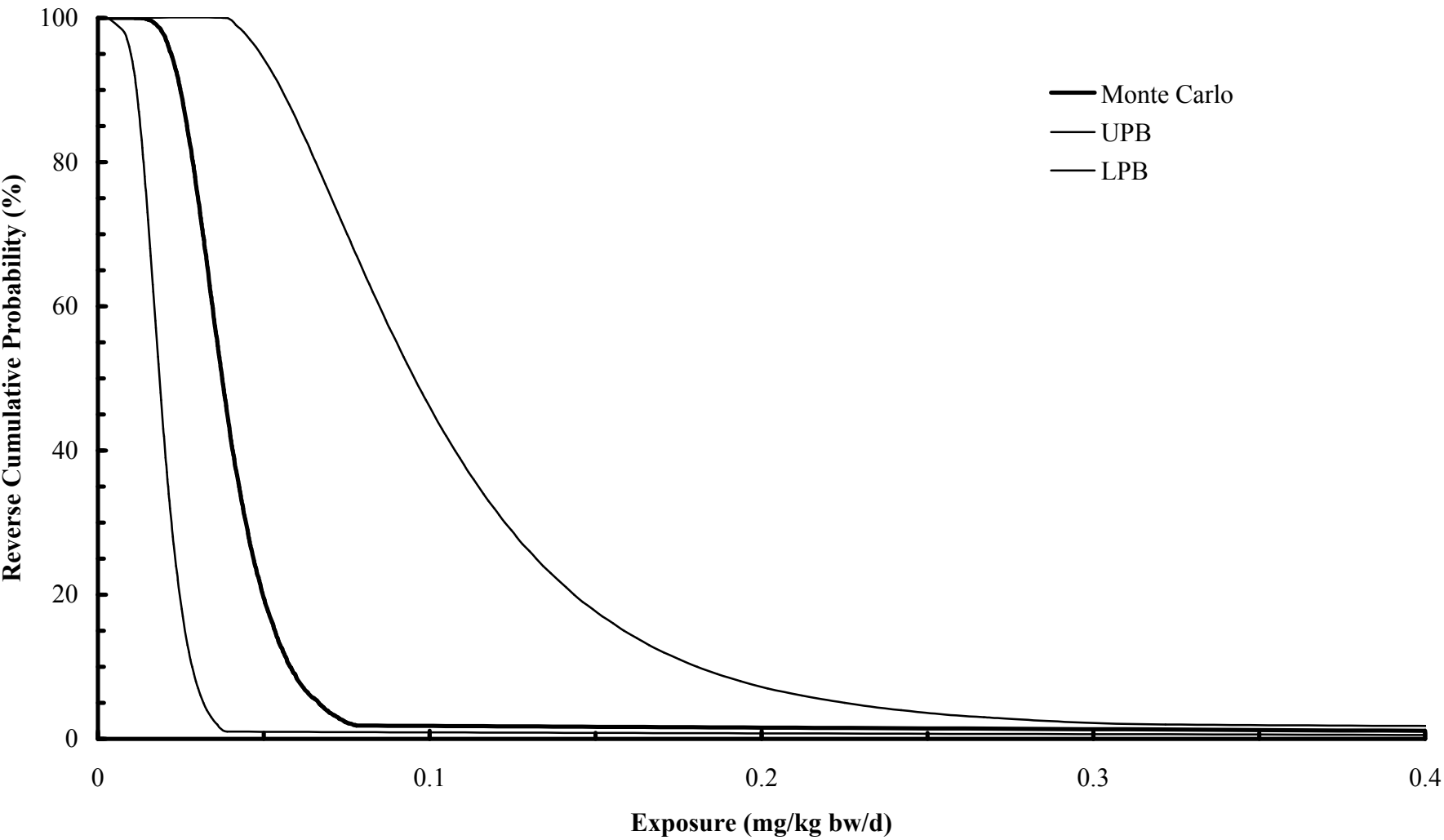
**Figure I2-29. Reverse cumulative probability distribution for average-sized piscivorous mammals exposed to selenium in reference areas (UPB and LPB are upper and lower probability bounds, respectively).**



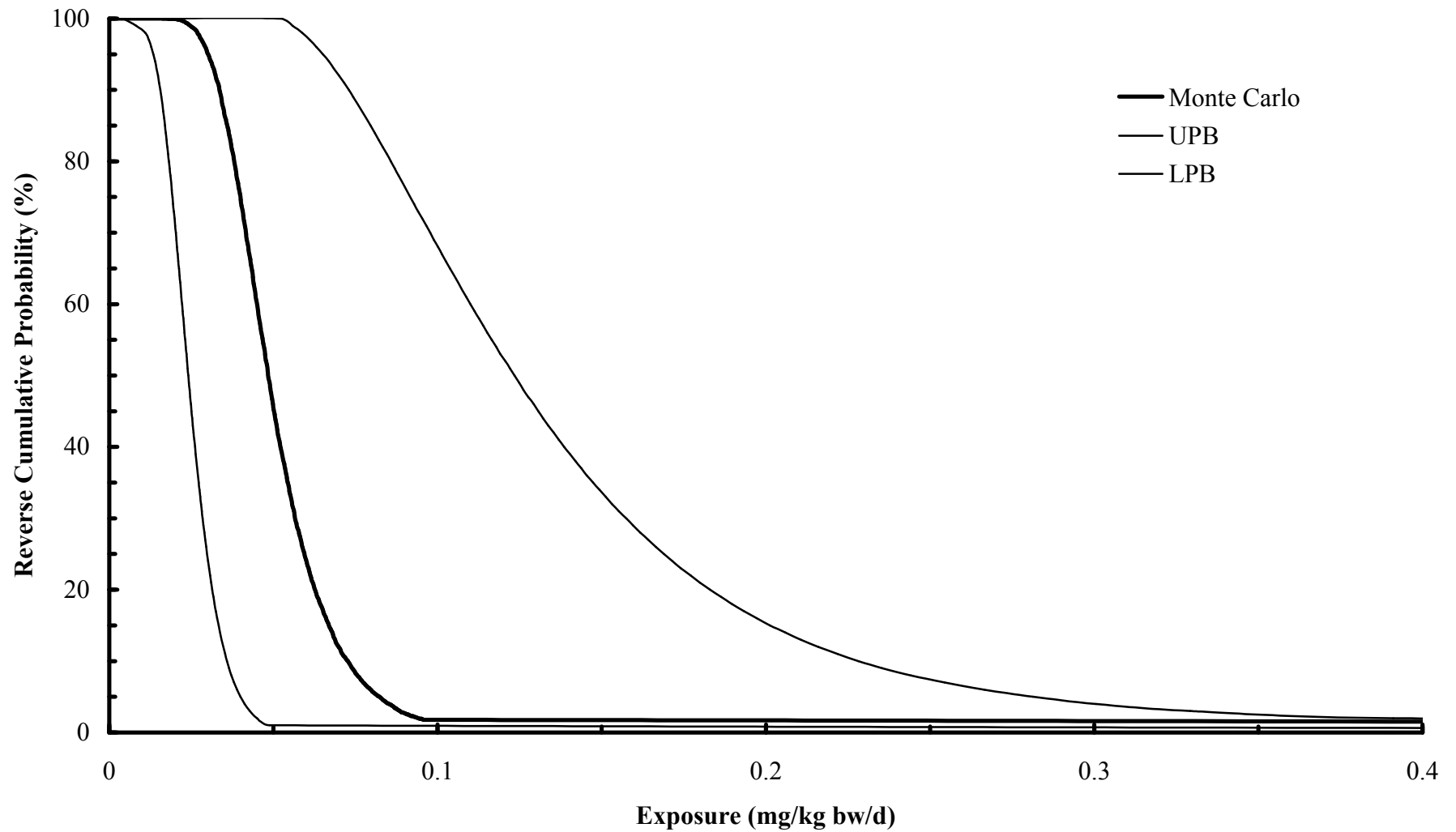
**Figure I2-30. Reverse cumulative probability distribution for small piscivorous mammals exposed to selenium in reference areas (UPB and LPB are upper and lower probability bounds, respectively).**



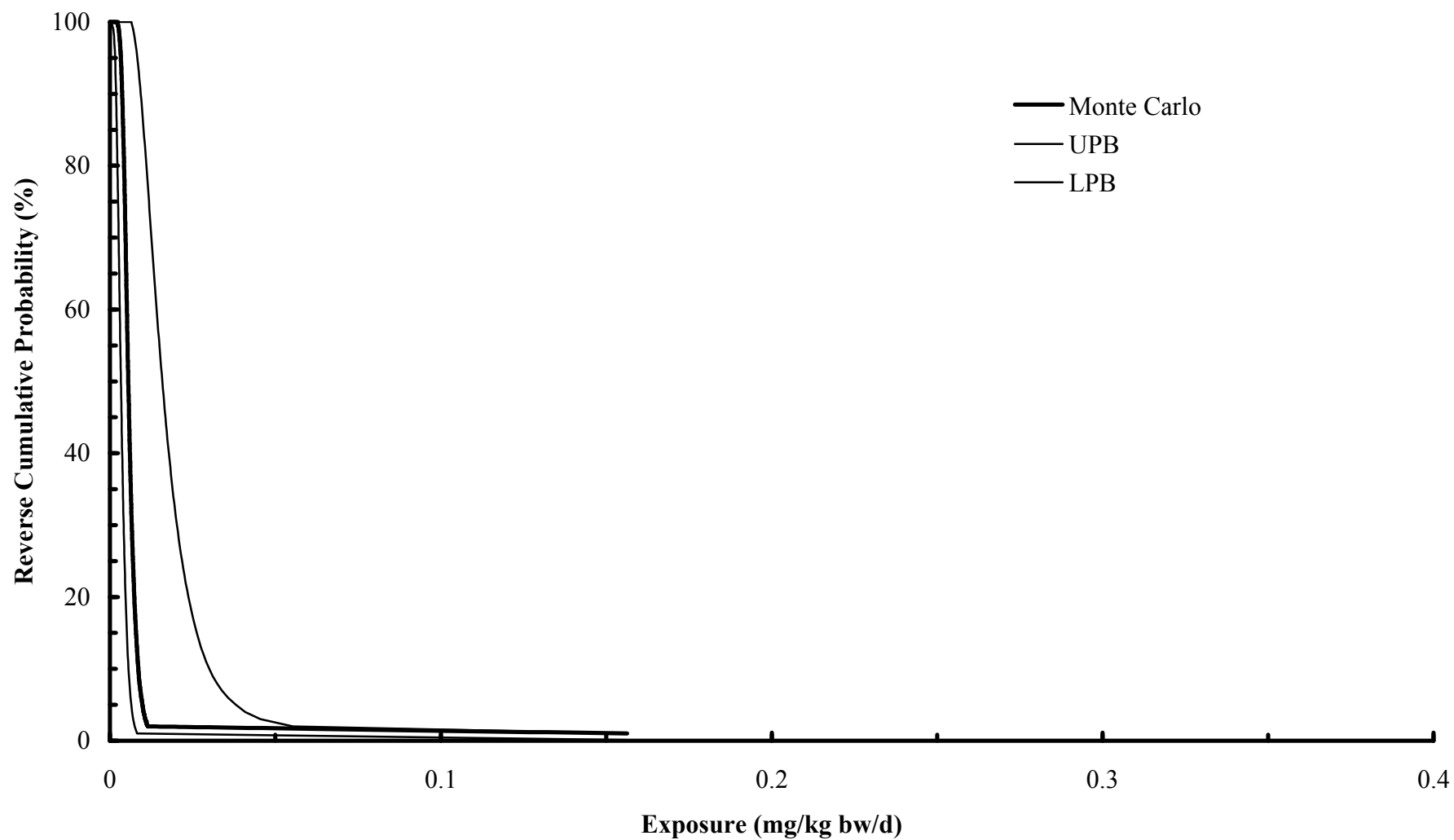
**Figure I2-31. Reverse cumulative probability distribution for average-sized piscivorous mammals exposed to total PCBs in Bayou d’Inde AOC (UPB and LPB are upper and lower probability bounds, respectively).**



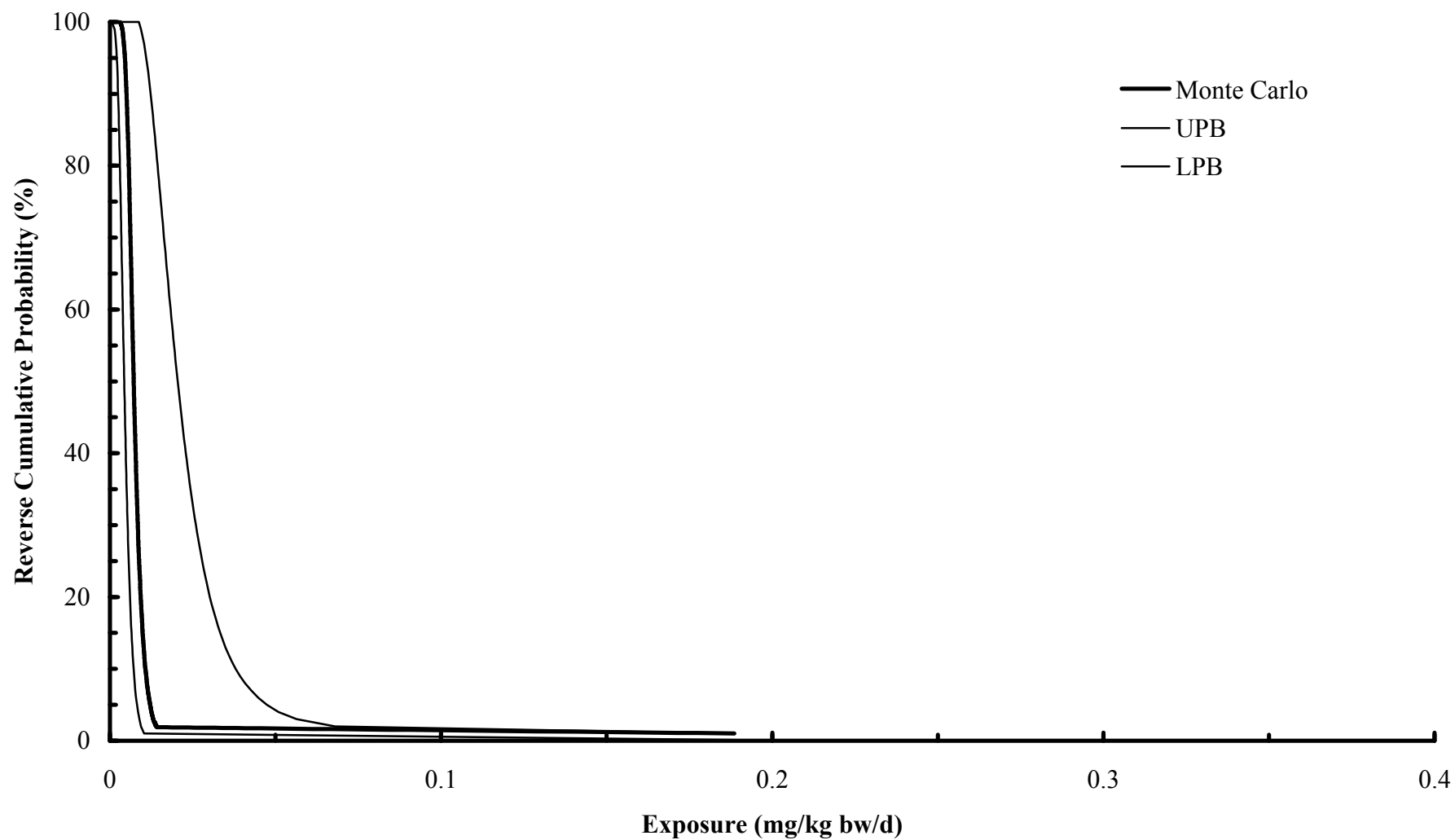
**Figure I2-32. Reverse cumulative probability distribution for small piscivorous mammals exposed to total PCBs in Bayou d'Inde AOC (UPB and LPB are upper and lower probability bounds, respectively).**



**Figure I2-33. Reverse cumulative probability distribution for average-sized piscivorous mammals exposed to total PCBs in reference areas (UPB and LPB are upper and lower probability bounds, respectively).**



**Figure I2-34. Reverse cumulative probability distribution for small piscivorous mammals exposed to total PCBs in reference areas (UPB and LPB are upper and lower probability bounds, respectively).**



**Figure I2-35. Risk function for average-sized female piscivorous mammals exposed to mercury in Bayou d'Inde AOC.**

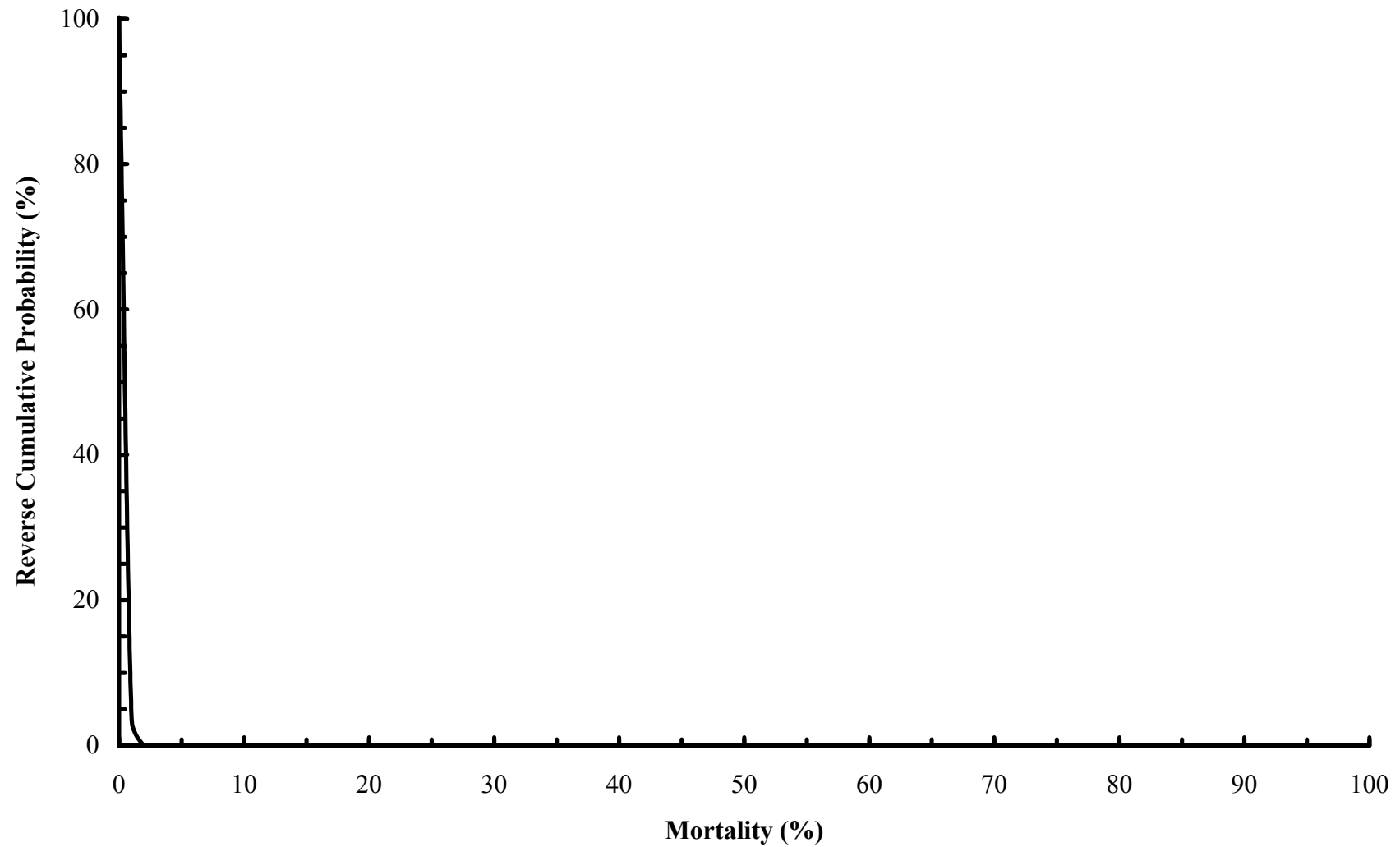
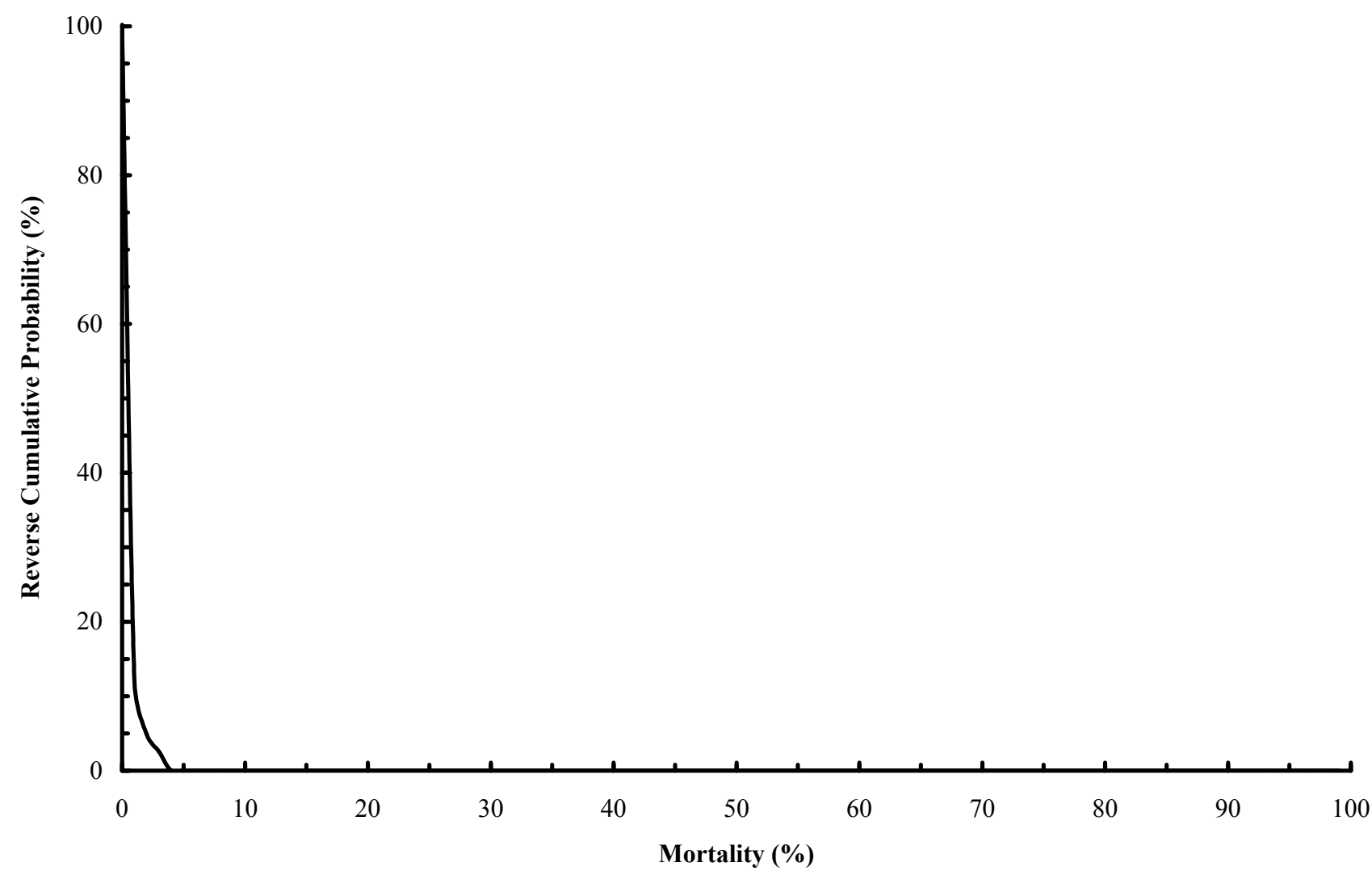
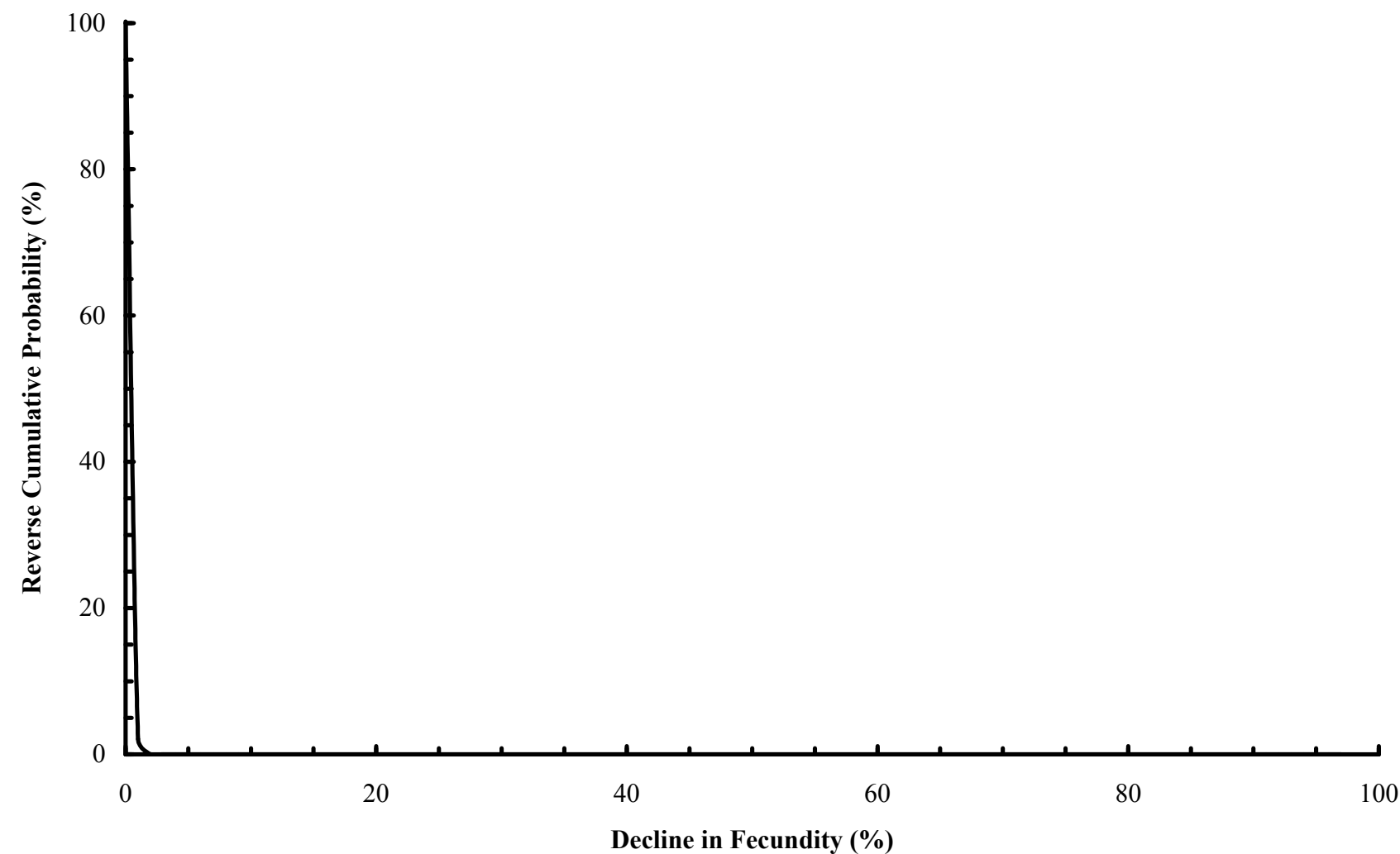


Figure I2-36. Risk function for small female piscivorous mammals exposed to mercury in Bayou d'Inde AOC.



**Figure I2-37. Risk function for average-sized piscivorous mammals exposed to TCDD and equivalents in Bayou d'Inde AOC.**



**Figure I2-38. Risk function for small piscivorous mammals exposed to TCDD and equivalents in Bayou d'Inde AOC.**

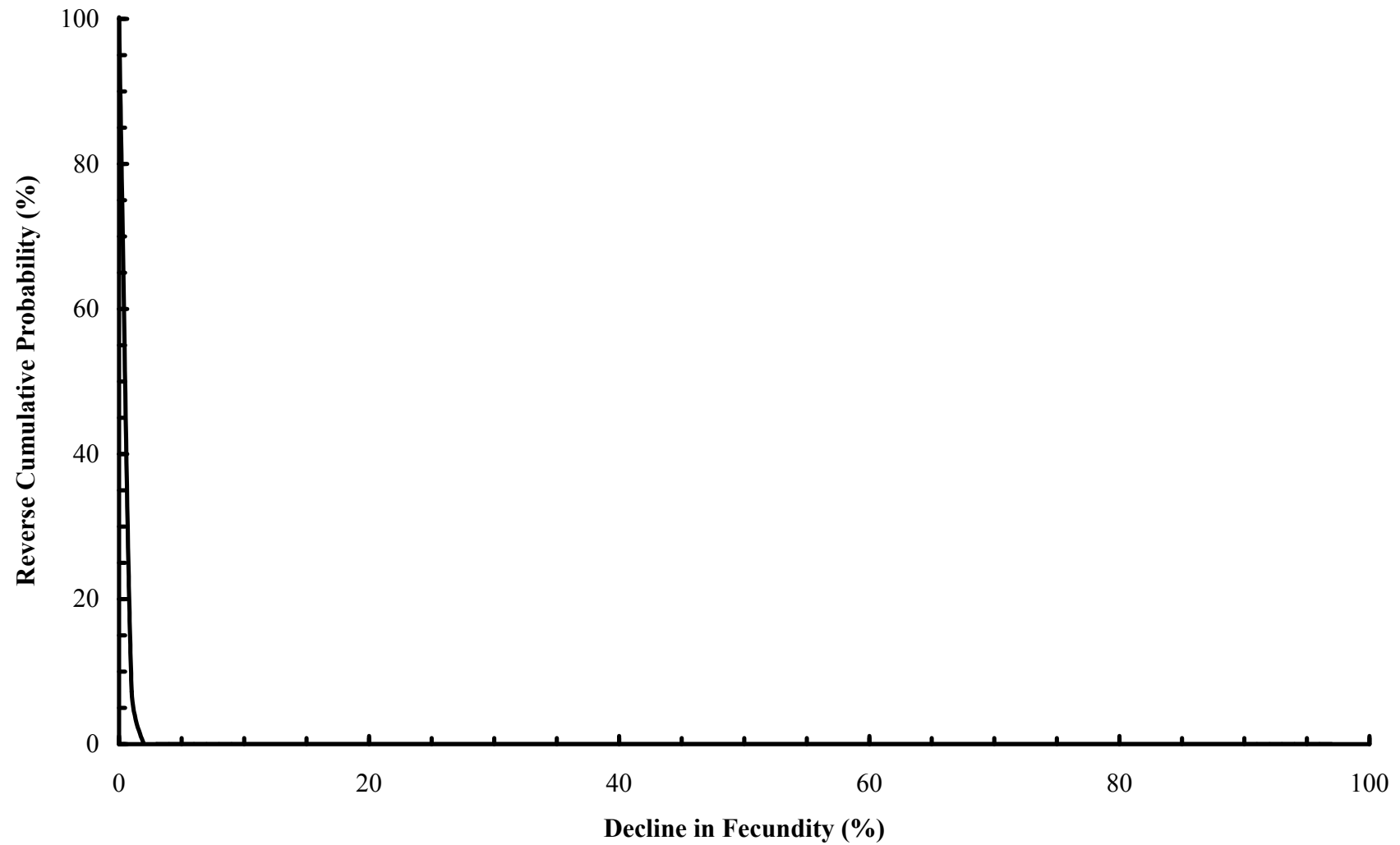


Figure I2-39. Risk function for average-sized piscivorous mammals exposed to total PCBs in Bayou d'Inde AOC.

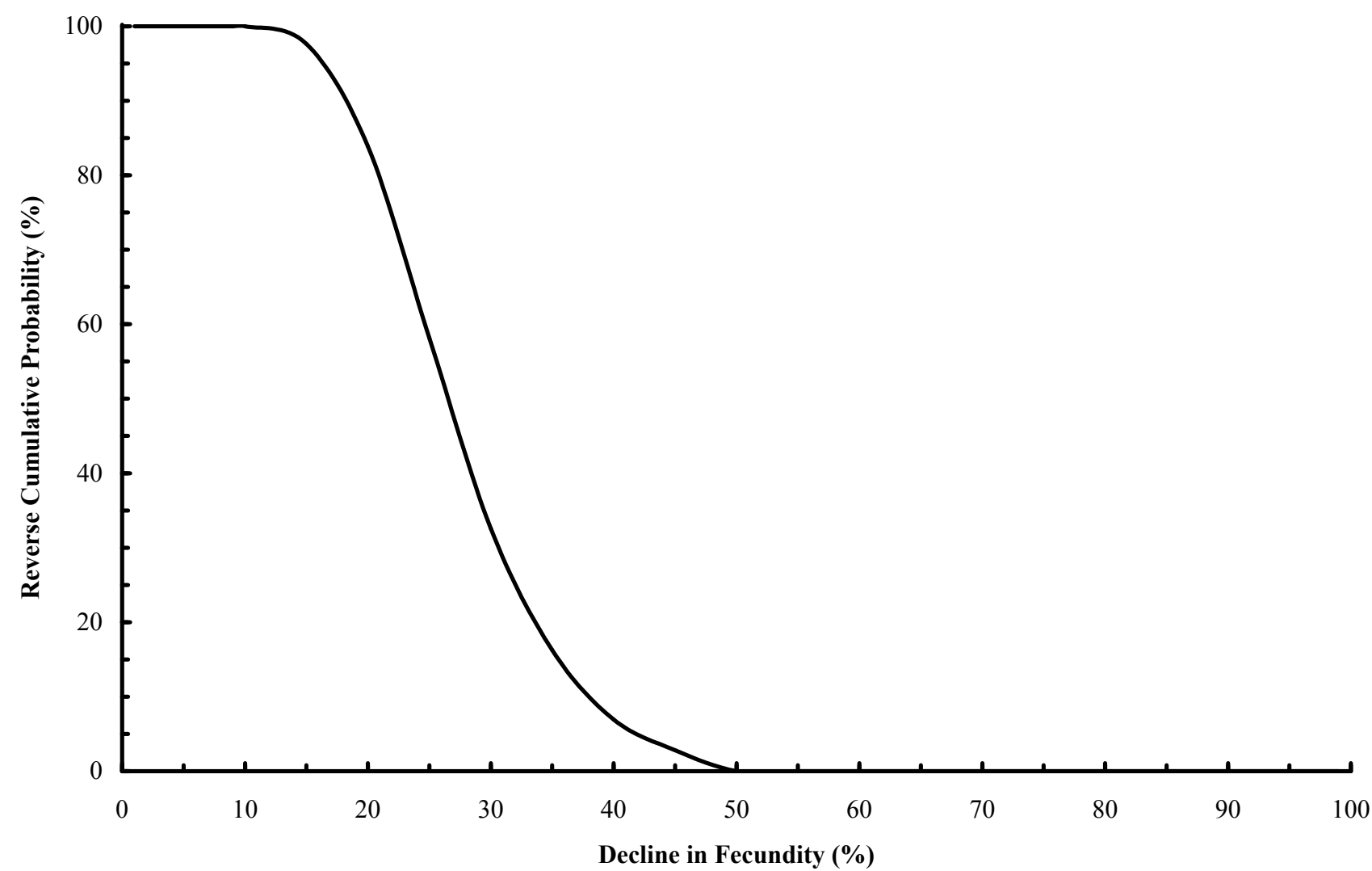
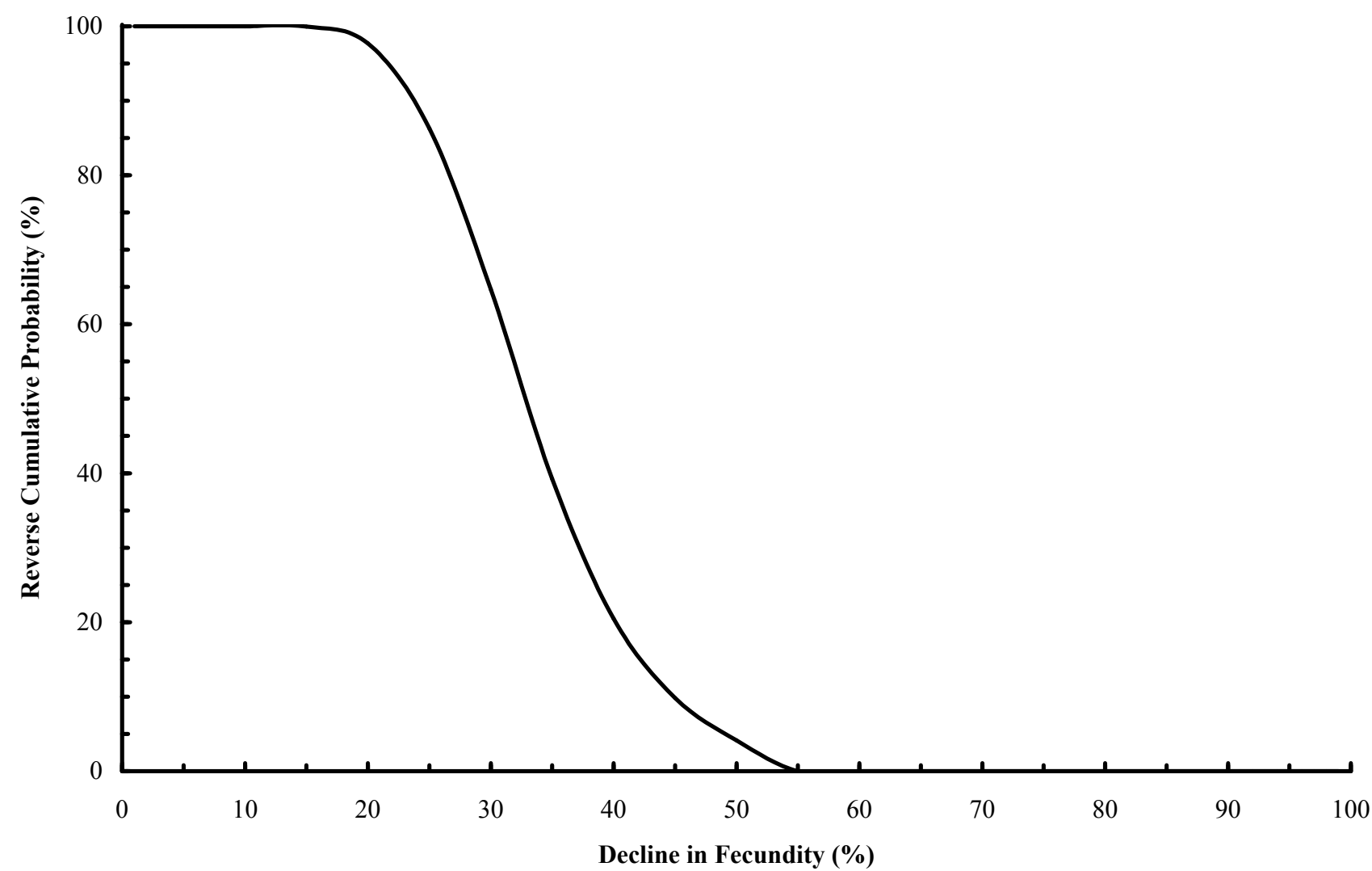
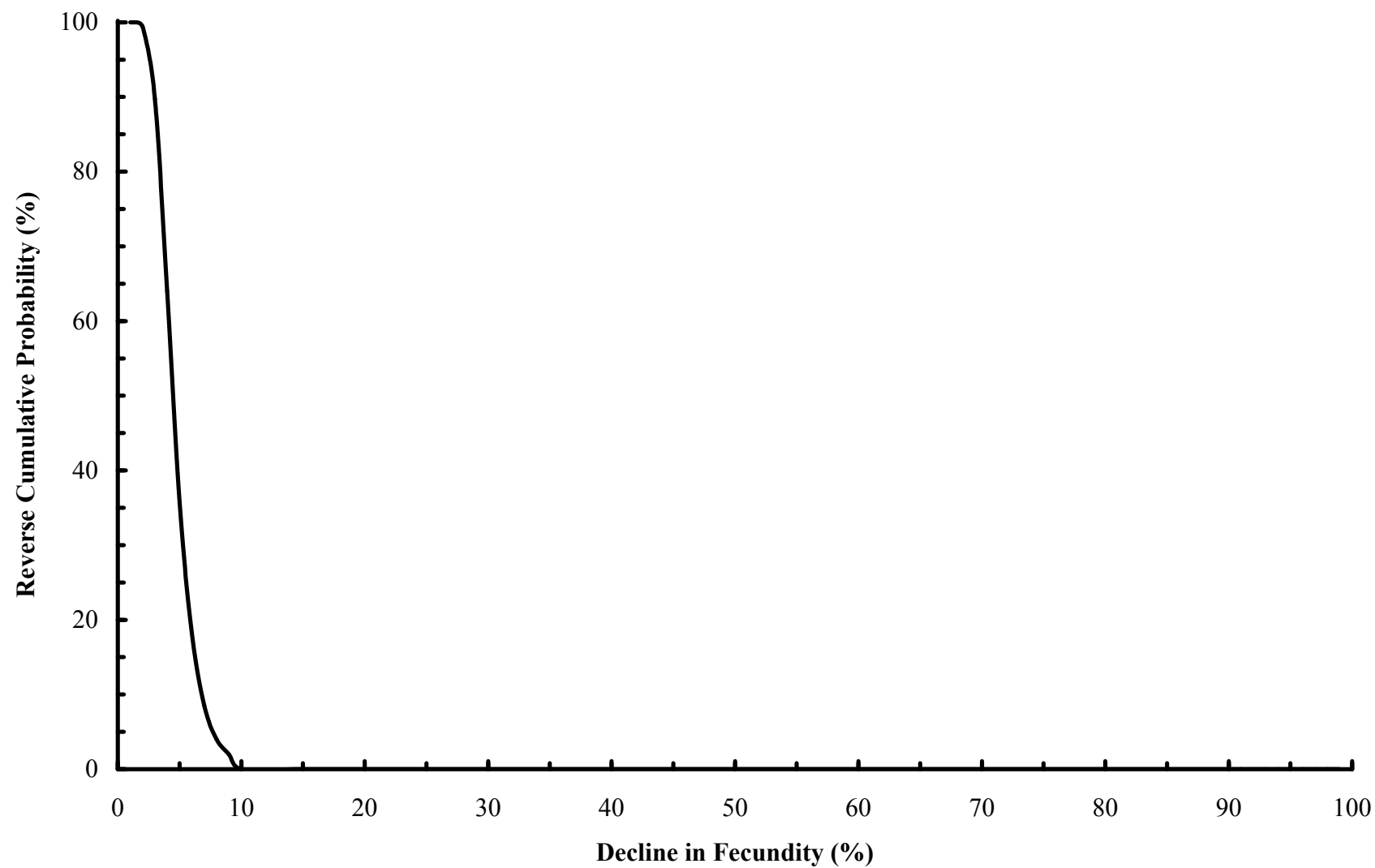


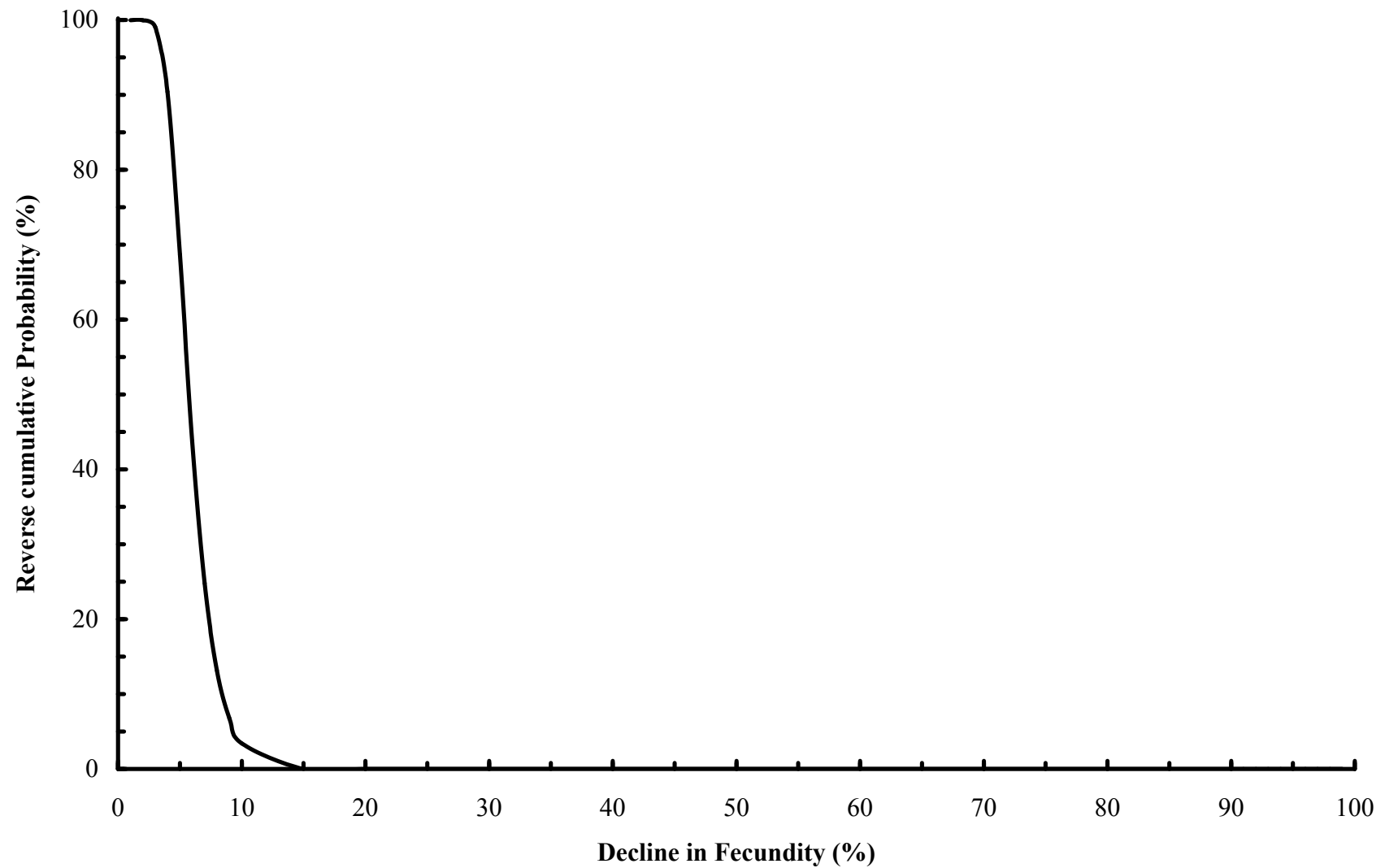
Figure I2-40. Risk function for small piscivorous mammals exposed to total PCBs in Bayou d'Inde AOC.



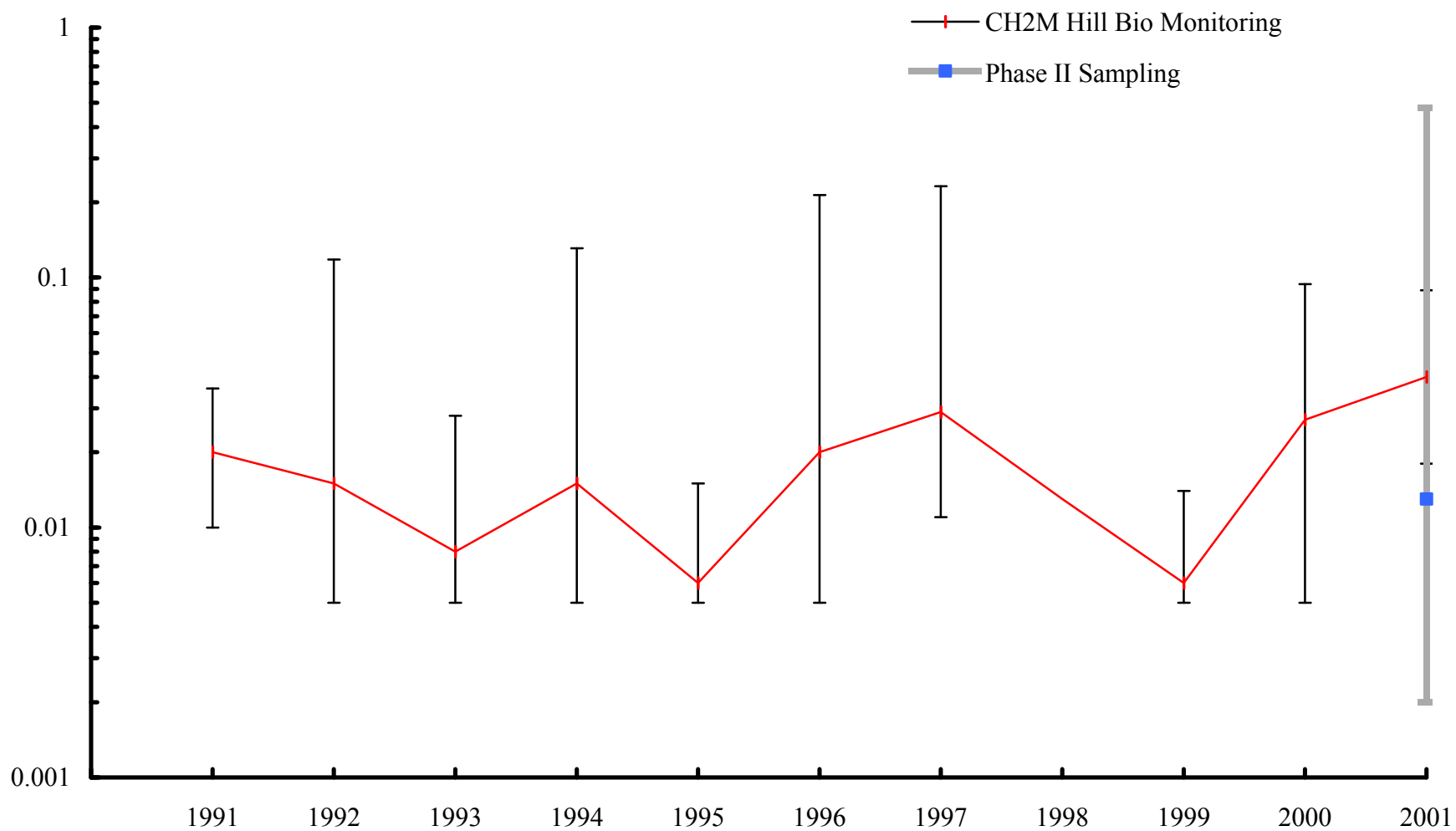
**Figure I2-41. Risk function for average-sized piscivorous mammals exposed to total PCBs in reference areas.**



**Figure I2-42. Risk function for small piscivorous mammals exposed to total PCBs in reference areas.**



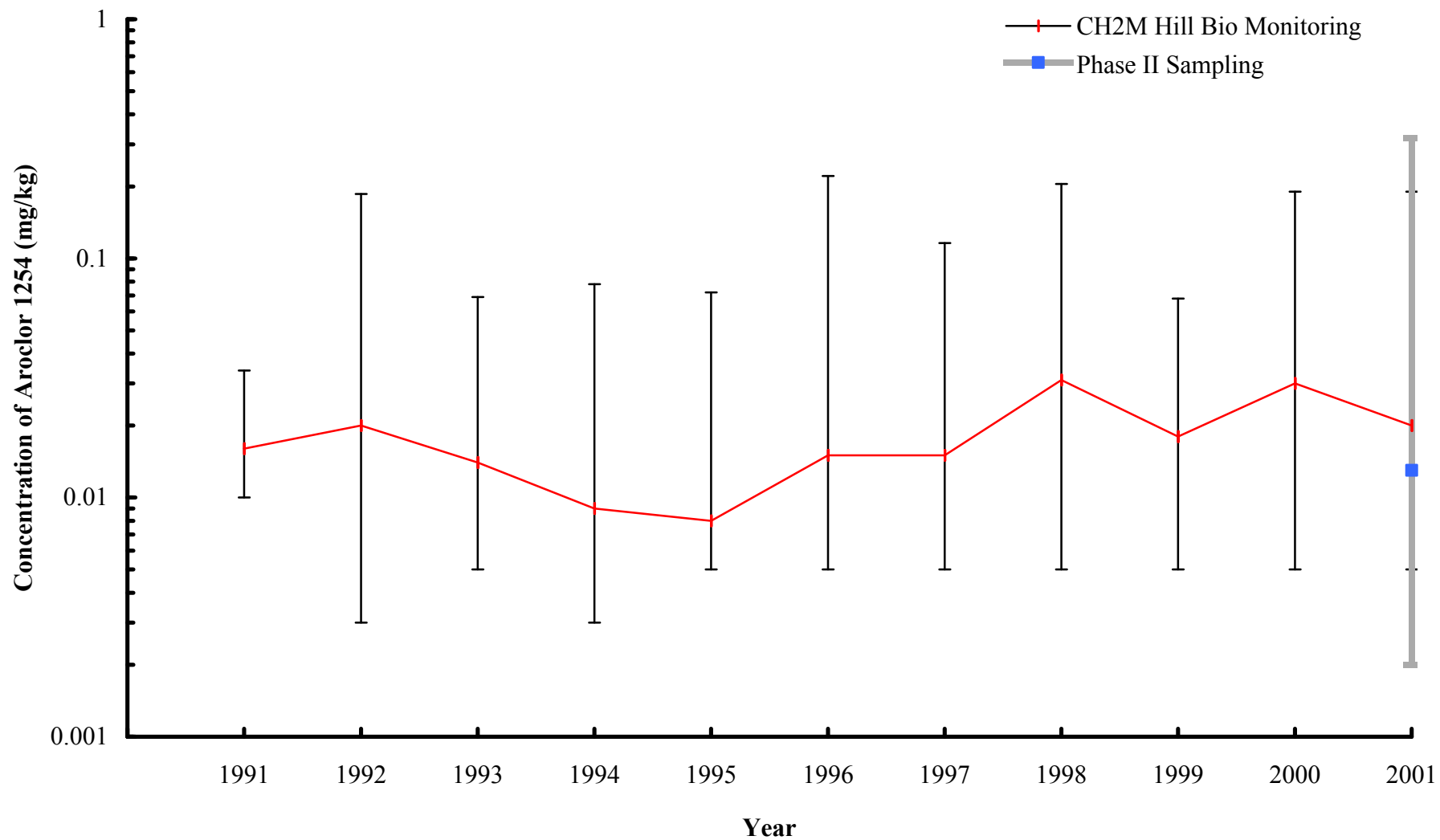
**Figure I2-43. Annual geometric mean concentration of Aroclor 1254 in fish fillet from the Upper Calcasieu River AOC (bars represent minimum and maximum concentrations).**



**Figure I2-44. Annual geometric mean concentration of Aroclor 1254 in fish fillet from Bayou d'Inde AOC (bars represent minimum and maximum concentrations).**



Figure I2-45. Annual geometric mean concentration of Aroclor 1254 in fish fillet from the Middle Calcasieu River AOC (bars represent minimum and maximum concentrations).



**Figure I2-46. Annual geometric mean concentration of Aroclor 1254 in fish fillet from reference areas of the Calcasieu Estuary (bars represent minimum and maximum concentrations).**

